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IX. REID'S BASE LINE

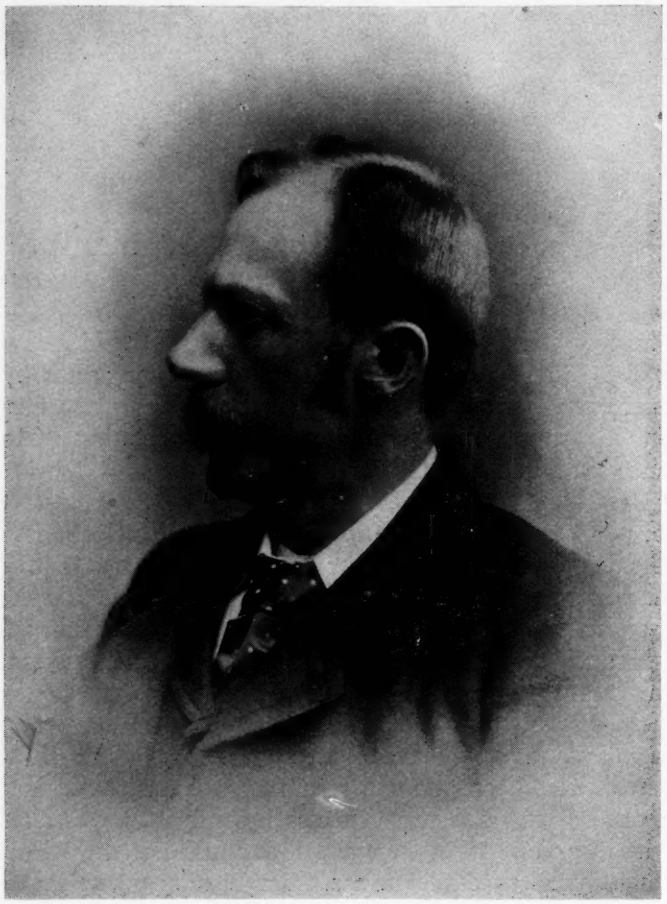
MUCH good work was being done on the brain in England between 1870 and 1890. Ferrier and Yeo were localizing the functions of the cortex by experimental methods; Schafer as an histologist was tracing the course of the fibres and tracts; Reid as an anatomist was giving surgeons a guide for operating; Macewen, Victor Horsley, Ballance, and Rickman Godlee were operating.

Professor Reid published his communication in *The Lancet* (1884, ii, 539) under the title, "Observations on the Relation of the Principal Fissures and Convolutions of the Cerebrum to the Outer Surface of the Scalp". He was then Lecturer on Anatomy in the Medical School of St. Thomas's Hospital, and is now Emeritus Regius Professor of Anatomy at Aberdeen, and Honorary Curator of the University Anthropological Museum, of which he was the Founder. A Reid Lectureship in Anthropology was founded in his honour at Aberdeen University in 1934, and the first lecture was given by him on December 3, 1934. The portrait which he very kindly sent at my request was taken in 1889, the year he left London for Aberdeen. He says in the article referred to above:—

"What I propose to do is to show that by taking large and easily felt landmarks on the head, and drawing from these certain lines, these lines will indicate accurately enough for all practical purposes the position of the principal sulci, and that by removing in any of these lines a piece of the scalp and skull an inch square, or by applying the one inch trephine to the skull, with the centre pin on the line, we can expose the fissure in any part of its course.

"The landmarks which can be easily felt on the outside of the scalp, and with reference to which lines can be drawn to indicate the position of the sulci are the glabella, or depression between the two nasal eminences, just above the root of the nose, external occipital protuberance, superior curved line of the occipital bone, parietal eminence, posterior border of the mastoid process, depression just in front of the external auditory meatus, external angular process of the frontal bone, frontal part of the temporal ridge and the supra-orbital notch. We shall also suppose that the Base line (*see Figure*), from which all perpendicular lines are drawn, runs through the lowest part of the infra-orbital margin and the middle of the external auditory meatus."

The line is not further defined in the text, but it soon passed into common use as 'Reid's base line' and was serviceable to several generations of surgeons as a rallying point when small trephines were used, osteoplastic flaps had not been thought of, and operations were limited to the cortex. It is now more of historical interest than of practical value. Reid continues :—



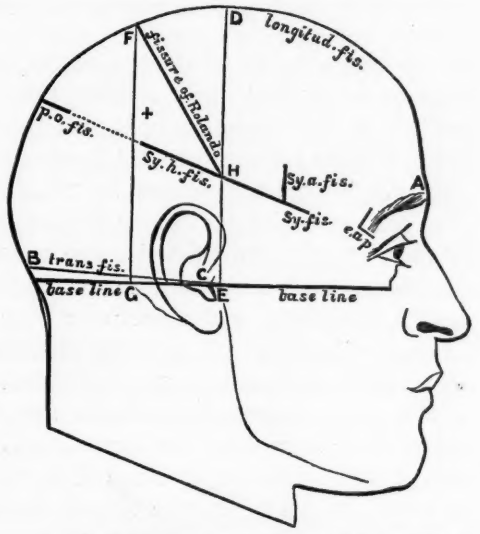
PROFESSOR REID

" *Transverse Fissure.*—This is indicated by drawing a line along the superior curved line of the occipital bone from the external occipital protuberance to the external auditory meatus—that is, along the line of junction of the head with the neck posteriorly.

" *Fissure of Sylvius.*—To find this draw a line from a point one inch and a quarter behind the external angular process of the frontal bone to a point three-quarters of an inch below the most prominent part of the parietal eminence. Measuring from before backwards, the first three-quarters of an inch of the line will represent the main fissure, and the rest of the line will indicate the horizontal

limb. The ascending limb will start from the posterior end of the line indicating the main fissure—that is, two inches behind and slightly above the external angular process—and run vertically upwards for about an inch (*see Figure*).

"*Fissure of Rolando.*—To find this, and at the same time the region of the ascending frontal and parietal convolutions, first indicate on the surface of the scalp the longitudinal fissure and the horizontal limb of the fissure of Sylvius, next, from the base line draw two perpendicular lines to the top of the cranium, one (DE) from the depression in front of the external auditory meatus, and another (FG) from the posterior border of the mastoid process at its root. We shall then have described on the surface of the head a four-sided figure, bounded above and below by the lines for the longitudinal fissure and horizontal limb of the fissure of Sylvius respectively, and in front and behind by the two perpendicular lines. If we now draw a diagonal line (FH) from the posterior superior angle to the anterior inferior angle of the space, this diagonal line will lie over the fissure of Rolando. In the majority of cases the fissure of Rolando does not run actually into the fissure of Sylvius, a convolution bridging across at that point; hence the lowest half inch or so of the line would cross this convolution. In those cases where the two fissures join, the fissure of Rolando would be indicated by the whole length of the line.



A, Glabella. B, External occipital protuberance. *e.a.p.*, External angular process of frontal. BC, Transverse fissure. AB, Longitudinal fissure. *Sy.fis.*, Sylvian fissure. *Sy.h.fis.*, Horizontal limb of fissure of Sylvius. *Sy.a.fis.*, Ascending limb of fissure of Sylvius. DE, Perpendicular line from depression in front of external auditory meatus to middle line of top of head. FG, Perpendicular line from posterior end of base of mastoid process to middle line of top of head. FH, Fissure of Rolando. *p.o.fis.*, Parieto-occipital fissure. +, Most prominent part of parietal eminence.

"*External Parieto-occipital Fissure.*—This fissure is more variable in its position than any of the other principal fissures of the cerebrum and consequently any indication of it on the surface of the scalp must be only approximately accurate. If we continue the line for the horizontal limb of the fissure of Sylvius onwards to the line indicating the longitudinal fissure we shall find that on trephining over the inner inch of this line we shall expose the external parieto-occipital fissure, or some part of it. In most cases we have seen the whole of the fissure in the opening so made, in others it showed itself in the posterior half of the opening, and in still fewer cases it was seen in the anterior half."

Having thus indicated the principal fissures Professor Reid proceeded to map out the areas of the frontal, parietal, temporo-sphenoidal, and occipital lobes with their chief sulci and convolutions as far as they are in relation with the outer surface of the scalp. He says that his descriptions are taken from a series of dissections made by himself in the post-mortem room of St. Thomas's Hospital. They were frequently tested and had been found to be accurate. The observations were intended to give a general idea of the surface of the cerebrum in relation to the scalp.

THE RADIOGRAPHY OF THE DUODENAL CAP

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In the examination of the duodenum it can be claimed that modern radiography is more exact and more reliable than either clinical or laboratory investigation. In fact it will reveal more than all the other methods put together. Time and again it gives information which can be obtained in no other way at present available. It is the only means by which incontestable evidence of an ulcer can be obtained, including its exact situation, and whether single or multiple. It can go further. It will show the size and condition of the ulcer and the presence of complications, such as penetration or stricture, the result of scarring changes from healed ulceration, and sometimes even the likelihood of impending perforation. Thus the findings will help the clinician to estimate the relative value of medical or surgical treatment in any individual case.

It is the common experience that there are many pitfalls in the radiographic diagnosis of lesions of the cap. The X-ray findings frequently lack the decisiveness which might be expected from an examination which is essentially objective in character. This is due almost entirely to a failure to employ an appropriate technique in the examination of the cap. Diagnostic accuracy is inextricably bound up with technique. Provided an appropriate technique is employed, there is no portion of the alimentary tract which can be so thoroughly investigated and recorded as the duodenal cap. The radiological diagnosis of duodenal ulcer can then be made with almost mathematical accuracy, for in the majority of cases the inner aspect of the cap can be shown on the film as clearly as in the resected specimen. Such a technique is the 'aimed exposure with dosed compression' technique, evolved by Professor H. H. Berg, of Hamburg.

In the early days technical difficulties in radiography prevented a satisfactory representation of the cap. As a result diagnosis was based on indirect sign-complexes in which changes in the function and form of the stomach played a predominant part. The first real advance towards the demonstration of direct radiological signs in the cap was the result of the investigations of Cole,[1] and George and Gerber,[2] who advocated the serial radiography of the cap. By correlating clinical, radiographic, operative, and pathological findings it was established that an unchanging contour deformity of the duodenal cap, in the majority of cases, spoke for duodenal ulcer. Akerlund[3] was, however, responsible for the greatest advance in the demonstration of the direct ulcer sign. By applying varying degrees of pressure ('dosed compression') to the cap under screen control, the optimum degree of pressure for the isolation and demonstration of the ulcer niche was determined. Single or serial radiographs were then made. Berg[4] crowned and completed this work by the introduction of the 'aimed exposure'.

TECHNIQUE

The object of the Berg technique is to present the details of the mucosal surface of the cap. In order to carry out the examination, some modification of the conventional screening apparatus is necessary. Some means must be provided by which varying degrees of compression (dosed compression) can be applied to allow the presentation of the mucosal relief. In addition the screening stand and power unit must be so arranged that a film can be exposed at any moment during the course of screening. The delay between the observation of the image on the screen and the recording of it on the film must not exceed a fraction of a second. Thus an aimed exposure is made. These aimed exposures are taken at the most varied angles and positions whenever there is any suspicion of abnormality. They provide a permanent record which can be studied at leisure and will often show details which even the most experienced examiner cannot detect on the screen. The ordinary vertical screening stand can be adapted for this work, but it will be found that an appropriately designed tilting couch facilitates the examination, especially so in those cases which are difficult in the erect position.

THE MUCOUS MEMBRANE

The cap differs from the other parts of the duodenum and from the remainder of the small intestine in that it is devoid of valvulæ conniventes. Although it was generally regarded as free from folds, Forssell,[5] in his investigations on the alimentary mucous membrane, showed that the cap presents a more or less typical mucosal pattern. As a rule, the folds are arranged longitudinally, continuous with the folds of the pyloric canal. On the radiogram the folds are represented by the translucent spaces, whilst the intervening hollows are seen filled with opaque medium. The folds are not visible when the cap is filled, but come into view when the superfluous opaque medium is displaced by pressure.

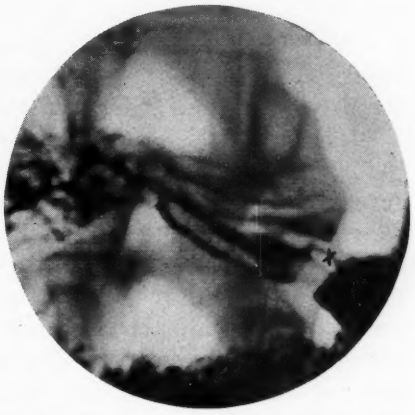

FIG. 472.—Normal mucosal relief pattern of duodenal cap. X, Pylorus.

The mucosal folds are not permanent anatomical structures, but vary during the process of digestion, so that a transverse arrangement of the folds, or a complicated rhomboidal, 'criss-crossed', or honeycomb pattern may be obtained. Thus a cap may show a varying mucosal pattern during the course of an examination. *Fig.* 472 shows a typical example of normal mucosal relief pattern.

The normal mucosal folds are soft and elastic and can be deformed and obliterated by stretching or by pressing over the cap. When a mild degree of pressure is applied, a broadening of the folds at first takes place and is associated with a corresponding narrowing of the intervening hollows. By increasing the amount of pressure the mucosal pattern can be completely obliterated. When, however, duodenitis or ulcer and its associated scarring changes pathologically

alters the mucous membrane, the mucosal folds are less easily influenced by pressure. Thus it is that pressure sufficient to obliterate normal mucosal folds brings into relief the details of the pathologically altered mucous membrane. The demonstration of a normal mucosal pattern in the cap definitely excludes the presence of an ulcer.

DUODENITIS

Amongst others, Judd and Nagel,[6] and Konjetzny[7] have shown that a chronic inflammation of the duodenal mucous membrane may occur even when no ulcer is present. This condition of duodenitis presents a clinical picture so similar to that of chronic duodenal ulcer that their clinical differentiation is seldom possible.

At operation the duodenum may appear normal, both to inspection and palpation. Often in the milder forms the only visible evidence on the serous surface of the duodenum is a localized area of hyperæmia with stippling, though the latter may only come into view when the serous surface is rubbed. In more advanced cases these signs are more marked and there may be a uniform narrowing of the duodenum for a distance of a few centimetres. There is, however, no evidence of scarring such as is found with ulcer. On palpation the wall of the duodenum seems normal and there is no induration such as is found with ulceration. When the duodenum is opened the mucous membrane is seen to be reddened and œdematous and may show minute superficial erosions. The lesions may be either localized or diffuse. As a rule the inflammatory process is limited to the mucosa. In some instances the submucosa shares in the inflammatory reaction, and the condition may be found, of course, in association with a chronic ulcer.

MacCarty[8] describes the pathological picture of duodenitis as that of cellular destruction with congestion, œdema, and migration of polymorphonuclear leucocytes, lymphocytes, and endothelial leucocytes.

The exact relationship between duodenitis and duodenal ulcer is not clear. Judd holds that duodenitis is not a stage from which true ulceration invariably develops, since the average duration of the symptoms of the two lesions is about the same. On the other hand, Konjetzny has found all stages from simple cellular infiltration of the mucosa and superficial erosions to large chronic, calloused ulcers in the same specimen. According to Kirklin,[9] the fundamental radiological characteristic of duodenitis is an extreme degree of irritability of a cap which is grossly deformed and contracted. Characteristic of such a deformed cap is its feathery and indistinct contour. The deformity, in addition to being much more extreme than with ulcer, is a changing one. Occasionally the cap may momentarily fill out and show a normal contour. The mucosal pattern is coarsely and irregularly reticular and is depicted as translucent islets lying in a dense network.

Berg has pointed out that direct changes can be demonstrated in the mucosal relief of the cap in cases of duodenitis. These changes are analogous with those which are found in the gastric mucosal picture in cases of hypertrophic gastritis. The latter condition is frequently to be found in association with duodenitis, and, though most commonly limited to the pyloric portion of the stomach, is sometimes generalized.

The most constant feature of a duodenitis is a veiling of the mucosal pattern by inflammatory exudate and a broadening and stiffening of the mucosal folds.

Not only are the folds broadened but so are the intervening hollows (*Fig.* 473). Duodenitis must not be diagnosed because of broadening of the mucosal folds unless they show changes in consistence. As the folds are examined under the screen they appear to have lost their elasticity, and give the impression that they have been starched. They can no longer be obliterated by stretching the duodenum and they are less easily deformed by pressure. Although the normal mucosal pattern may be retained, yet the folds often show abnormal direction (*Fig.* 474). Frequently deposits of the opaque medium may be retained between the swollen and abnormally directed folds and such deposits may simulate and be difficult to differentiate from a true niche. As a rule these pseudo-niches are larger and more irregular than a true ulcer niche, and do not fulfil the radiological criteria of niche formation when examined in both *en face* and profile views. Further, it is characteristic of such deposits that they are inconstant in situation during the same and also at subsequent examinations. These changes are most commonly seen in the

FIG. 473.—Duodenitis. Aimed exposure with dosed compression showing stiffened and broadened mucosal folds. X, Pylorus.

FIG. 474.—Duodenitis. Showing swollen mucosal folds with irregular pattern. X Pylorus.

cap, but similar changes may be present in the descending portion of the duodenum. Berg, however, points out that it is uncommon to find them beyond the papilla of Vater—in other words, they are localized to the common ulcer sites.

Dense adhesions and periduodenal tumour-mass formations such as occur with penetrated ulcers are not found in duodenitis. Nevertheless, as the result of fine connective-tissue deposit on the serous surface of the duodenum, fine filamentous adhesions to the omentum, gall-bladder, and other organs may form. Such adhesions may produce no deformity of the contour, nor diminish the mobility of the duodenum, and consequently may escape radiographic recognition. When, however, adhesion takes place between the gall-bladder and the duodenum a characteristic deformity is often produced. Normally in the oblique positions, the junction of the cap and the descending portion of the duodenum is seen as a nicely rounded curve. When adhesion occurs between the gall-bladder and duodenum, this rounded curve is replaced by a persistent, acute angulation (*Fig.* 475A, B). In these cases cholecystographic examination will often show that the gall-bladder is drawn towards the mid-line, but its contour is normal, a good concentration of

the dye is obtained, and there is a normal contractile response to a fatty meal. Occasionally an adhesion will produce a small boss of the duodenal contour in the profile view and so simulate a niche (*Fig.* 475C). The differentiation can be made

FIG. 475.—Acute angulation at junction of cap and descending limb of duodenum, the result of adhesion to the gall-bladder. A, Confirmed at operation. B, Upper arrow indicates site of adhesion. Lower arrows point to ulcer niche on anterior wall. At operation adhesion confirmed. Scar on anterior wall of cap. C, Duodenal boss due to adhesion. Note absence of concave indentation of adjacent profile. X, Pylorus.

because the concave indentation of the profile on either side of the boss is lacking. This concave indentation, as has been stressed by Berg, is an essential accompaniment of a true niche.

THE NICHE

The niche is pathognomonic of an ulcer in the duodenum as in the stomach. It is the only absolute evidence of loss of substance from ulceration, and with proper technique it is the most constant demonstrable sign. Whilst some recent publications emphasize the rarity of the direct sign—the niche—in the duodenum, others do not even mention it. The failure to realize the frequency and importance of the niche can only be due to failure in radiographic technique.

The conditions for the demonstration of the niche are particularly favourable, since, with few exceptions, ulceration in the duodenum is confined to the cap. The area to be examined is relatively small, and in the majority of cases every aspect of it can be thoroughly investigated and recorded. Whenever possible, the presence of a niche should be confirmed in two planes. Usually this can be done. Sometimes, however, it may be difficult and in a few cases impossible—for example, when there are extensive adhesions or in adipose individuals with a high-lying steer-horn stomach.

A niche has essentially two components : the crater filled with opaque medium, and the defects produced in the duodenal lumen by the reactive tissue changes in the immediate vicinity of the ulcer. Berg has laid down certain criteria to which a niche must conform. These criteria are absolute, since they can be interpreted and confirmed in terms of the pathological changes which occur with ulceration.

The *en face* niche—that is, the niche as viewed from its front—is seen most often when the patient is examined in the first or right anterior oblique position. The niche is usually hidden or is indistinct when the cap is distended, but can be isolated and recognized by applying a suitable degree of compression (*Figs.* 476, 477). Such niches as are seen only after compression are termed by Berg ' relief ' niches. In the ulcer of comparatively short duration the niche is represented by a dense, circular or oval, well-defined opaque deposit surrounded by a translucent halo, or, as Berg calls it, a ' ring wall ' (*Figs.* 476B, 477B). This ring wall is produced by the œdematous mucous membrane surrounding the ulcer and projecting into the duodenal lumen. In the more chronic ulcer the niche is usually smaller, and the ring wall cannot generally be seen in this view. Distinct folds of mucous membrane will, however, be seen converging in a star-like manner towards the crater (*Fig.* 478). This convergence of the mucosal folds is the result of the fibrosis and reactive contraction which accompanies the chronic ulcer.

Since in the majority of cases ulceration in the cap is found on the antero-lateral and postero-medial surfaces, the profile niche is best shown by examining the patient in the second or left anterior oblique position. The characteristic—indeed, the essential—feature of the profile niche is a projection from the contour with a concave indentation of the adjacent profile on either side (*Fig.* 479). In the recent ulcer this concave indentation is produced by the swollen mucous membrane surrounding the ulcer and corresponds to the ring wall of the *en face* view when seen at right angles (*Fig.* 480). In the chronic ulcer it is produced partly by swollen mucous membrane, partly by infiltrative thickening of the wall surrounding the ulcer, and partly as the result of scar-tissue contraction in the wall.

It is important to recognize when an ulcer has penetrated into a neighbouring organ. If one judges by the usual clinical standards—disappearance of symptoms for months or even years, and the absence of occult blood from the stools—penetrated ulcers may show an apparent cure by medical means. It can be shown radiographically, however, that such ulcers are invariably quite refractory to medical treatment.

Normally the duodenal cap can be freely moved on manipulation. When an ulcer has penetrated into a neighbouring organ the cap becomes fixed at this point, and such fixation can be determined by palpation during the screen examination. If the pancreas has been penetrated the posterior wall of the duodenum becomes

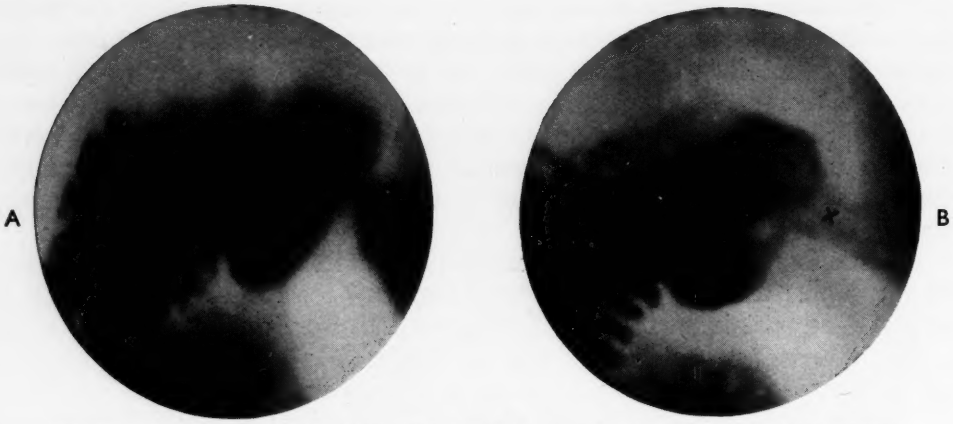

FIG. 476.—A, Aimed exposure. Faint shadow of niche. B, Aimed exposure with dosed compression. Niche with ring wall, due to swollen mucous membrane. X, Pylorus.

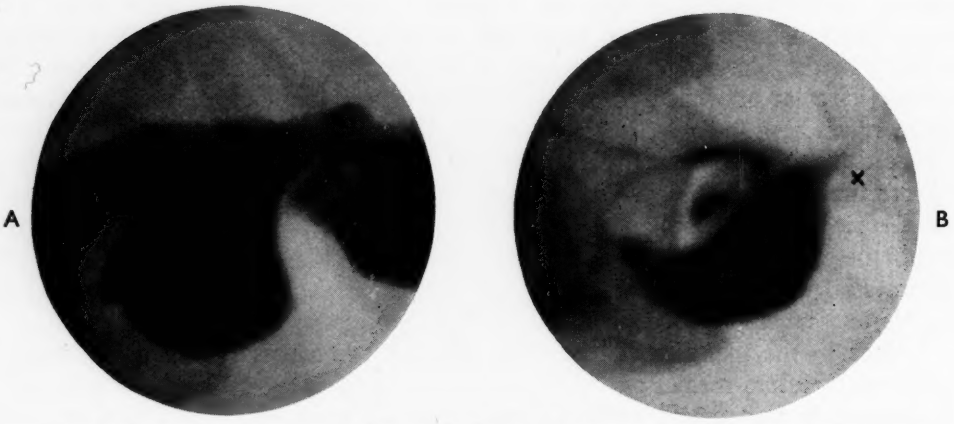

FIG. 477.—A, Aimed exposure. Niche with ring wall. B, Aimed exposure with dosed compression. Same case. X, Pylorus.

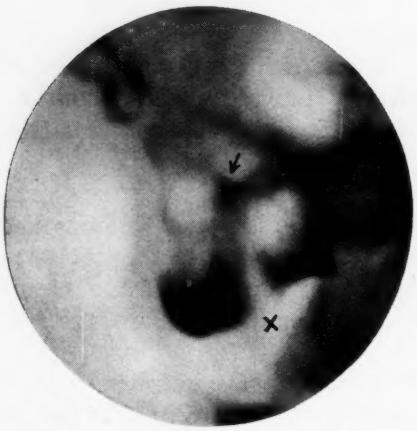

FIG. 478.—*En face* niche with star-like convergence of mucosal folds. X, Pylorus.

FIG. 479. — Profile niche, posterior wall. Concave indentation of adjacent profile. X, Pylorus.

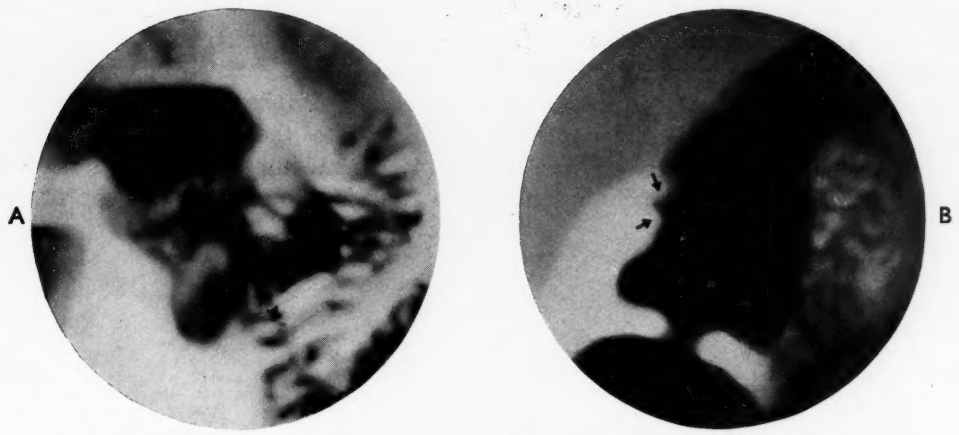

FIG. 480.—A, *En face* niche with ring wall. B, Profile view. Niche on anterior wall. Ring wall of *en face* niche shows as concave indentation of profile on either side of ulcer. X, Pylorus.

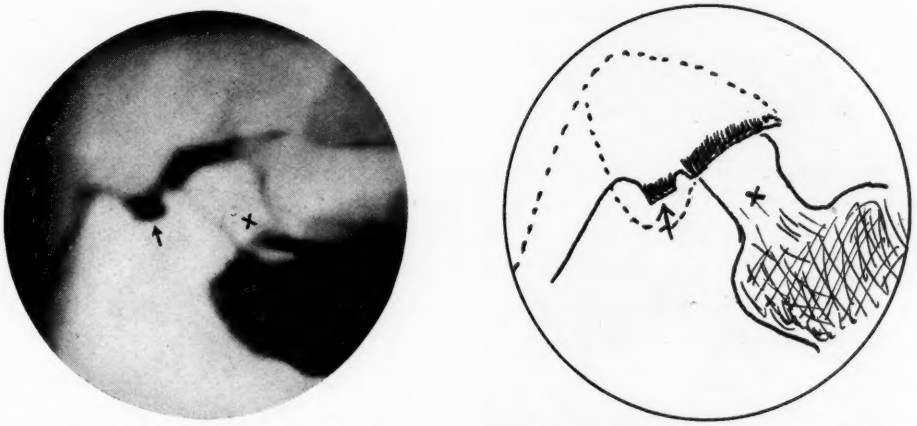

FIG. 481.—Posterior wall ulcer penetrated into pancreas. V-shaped deformity with niche. X, Pylorus. (Confirmed at operation.)

FIG. 482.—Posterior wall ulcer penetrated into pancreas. V-shaped deformity with niche. X, Pylorus. (Confirmed at operation.)

V-shaped when 'put on the stretch', and an ulcer niche will be found at the junction of the limbs of the V (*Figs.* 481, 482). Sometimes an accessory pocket with a communicating channel is seen outside the duodenal lumen (*Fig.* 483A). Occasionally one can show mucosal folds bent sharply away from the normal longitudinal direction and pointing into the channel (*Fig.* 483A). In the usual postero-anterior view the cap is seen foreshortened on account of its peculiar lie. Normally when the patient is placed in the right or left

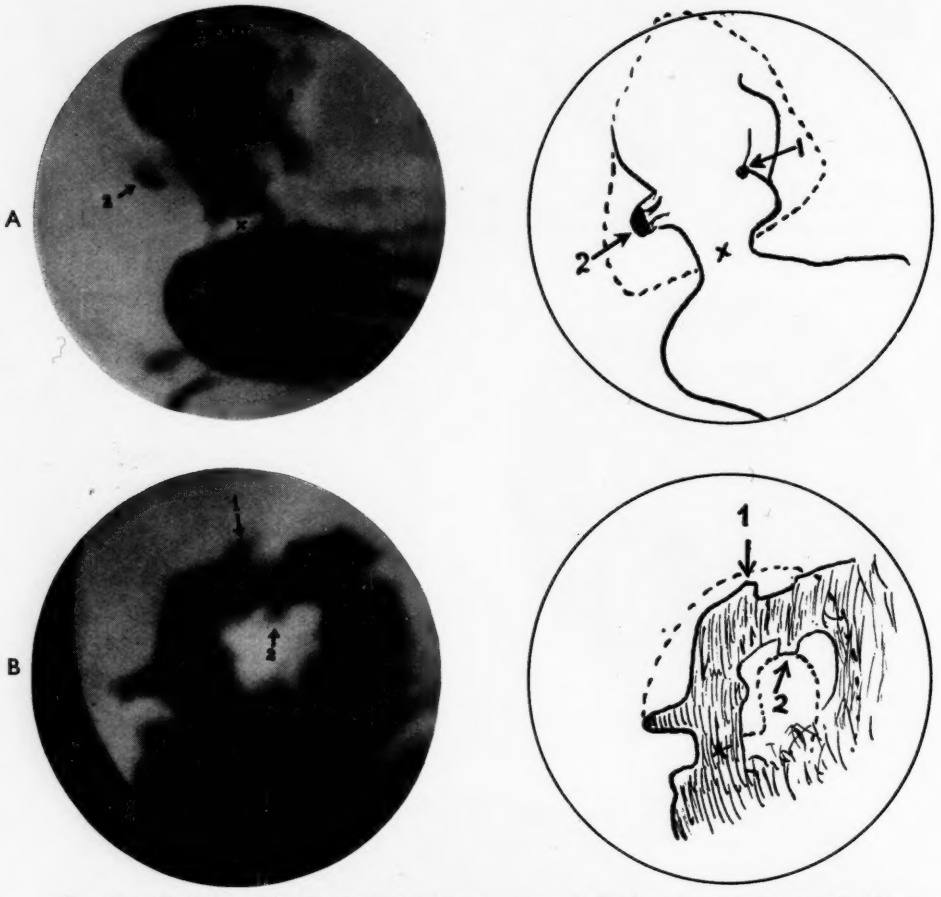

FIG. 483.—Posterior wall ulcer penetrated into pancreas. A, *En face* view. 2, Penetrated ulcer on posterior wall, showing accessory pocket and divergence of mucosal folds. 1, Anterior wall ulcer. B, Profile view of niches. V-shaped deformity of posterior wall with niche. Cap foreshortened in this view on account of fixation. X, Pylorus. (Confirmed at operation.)

anterior oblique positions the foreshortening disappears. If, however, the duodenum is fixed as the result of the penetration of an ulcer, the foreshortening persists in all positions.

The characteristics of a penetrating ulcer[10] may be summarized: (1) Fixation of cap; (2) V-shaped deformity with niche; (3) Accessory pocket; (4) Divergence of mucosal folds into communicating channel; and (5) Foreshortening. Although these characteristics may not be seen in every case, yet, if several are present, penetration may be deduced.

Multiple ulcers are common. Usually they are the 'kissing' ulcers of Moynihan. In the *en face* view the niches may be superimposed (*Fig.* 484A), but by turning the patient slightly, the two niches can be isolated (*Fig.* 484B).

FIG. 484.—'Kissing' ulcers. A, *En face* view—niches superimposed. B, *En face* view—niches isolated by turning patient slightly. C, Profile view of niches. X, Pylorus. (Confirmed at operation.)

FIG. 485.—Two ulcers in cap. Profile view. 1, Recent ulcer on anterior wall. 2, Chronic ulcer in distal portion of cap on posterior wall. X, Pylorus.

Confirmation of the niches is, of course, obtained in the profile view (*Fig.* 484C). The niches may be situated at different levels (*Fig.* 485), and three or more niches may be isolated in a cap (*Fig.* 486).

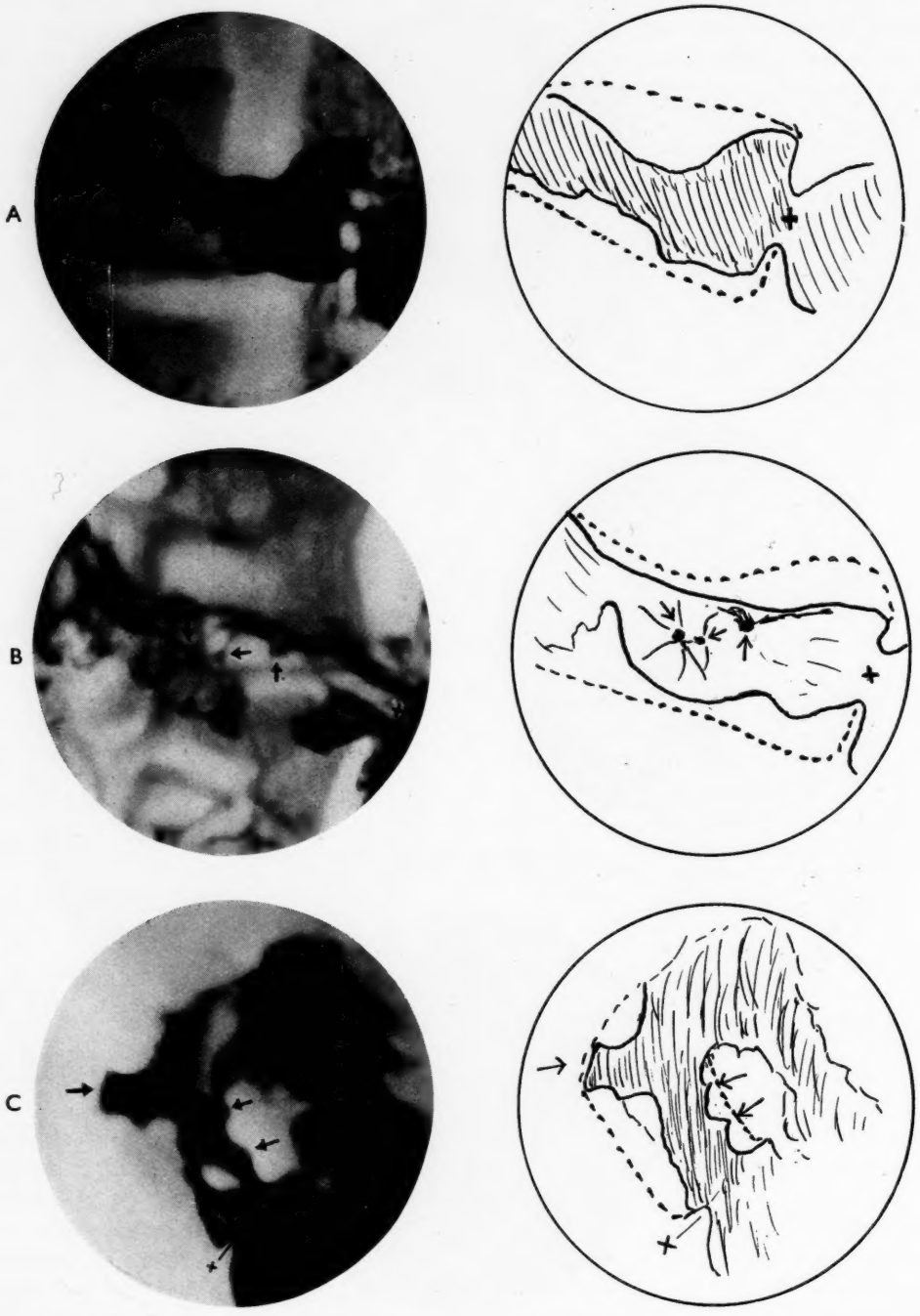

FIG. 486.—A, Aimed exposure showing deformed cap. B, Aimed exposure with dosed compression showing three niches *en face*. C, Profile view of niches. X, Pylorus.

Despite advances in technique, it must not be thought that every duodenal ulcer can be detected by X rays. Obviously, small shallow ulcers which do not retain the opaque medium will defy radiographic recognition. Under certain conditions even relatively large ulcers may be difficult to show. Berg holds that after a recent hæmorrhage a crater may be filled with blood-clot. The crater may also be blocked by mucus or by food remnants. In such a case the niche will be seen by giving a second meal immediately after the first has left the stomach and has exerted a cleansing effect upon the mucous membrane. Forssell holds that the 'autoplastik' of the mucous membrane may temporarily seal off the ulcer crater

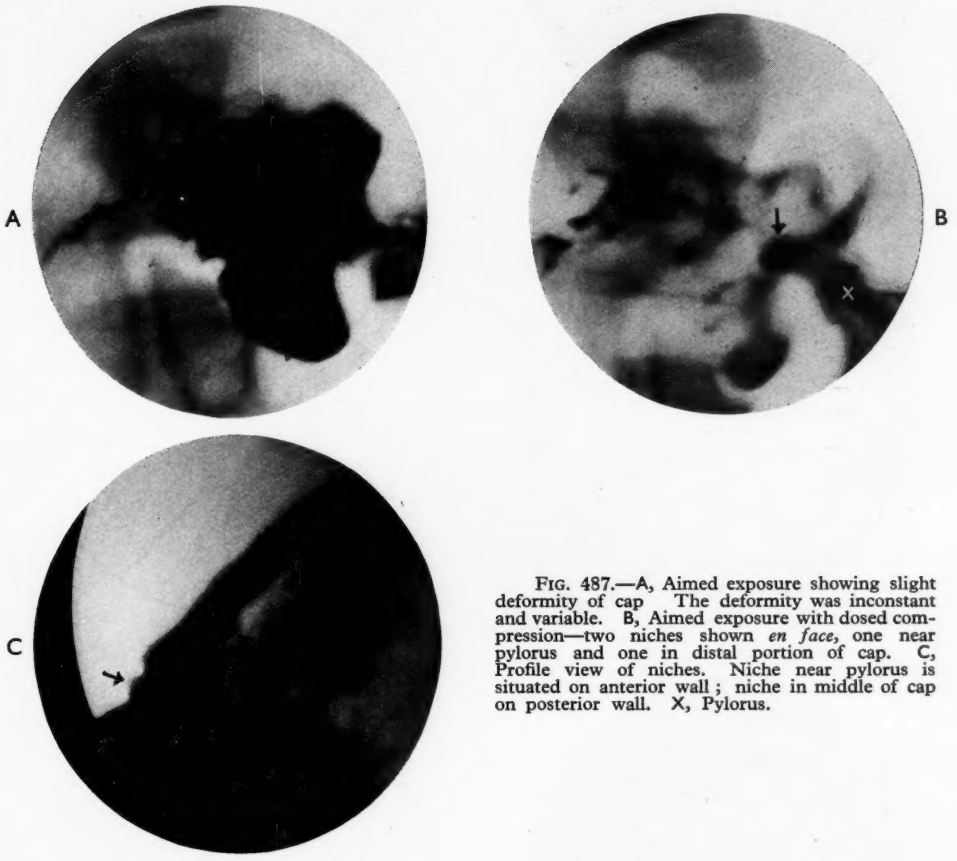

FIG. 487.—A, Aimed exposure showing slight deformity of cap The deformity was inconstant and variable. B, Aimed exposure with dosed compression—two niches shown *en face*, one near pylorus and one in distal portion of cap. C, Profile view of niches. Niche near pylorus is situated on anterior wall; niche in middle of cap on posterior wall. X, Pylorus.

from the duodenal lumen. Thorough palpation must always be employed so as to force the opaque medium into the crater.

A criticism which is often directed against the compression techniques of both Berg and Akerlund is that, by compression, deposits can be isolated and will be mistaken for ulcer-niches when, in fact, no ulcer exists. This will only occur if the fundamentals of technique and interpretation are not completely mastered. Provided a niche is only diagnosed when the strict criteria laid down by Berg are satisfied, the danger of diagnosing a non-existent ulcer is negligible. This has been proved beyond all doubt by the careful operative control of the radiographic findings.

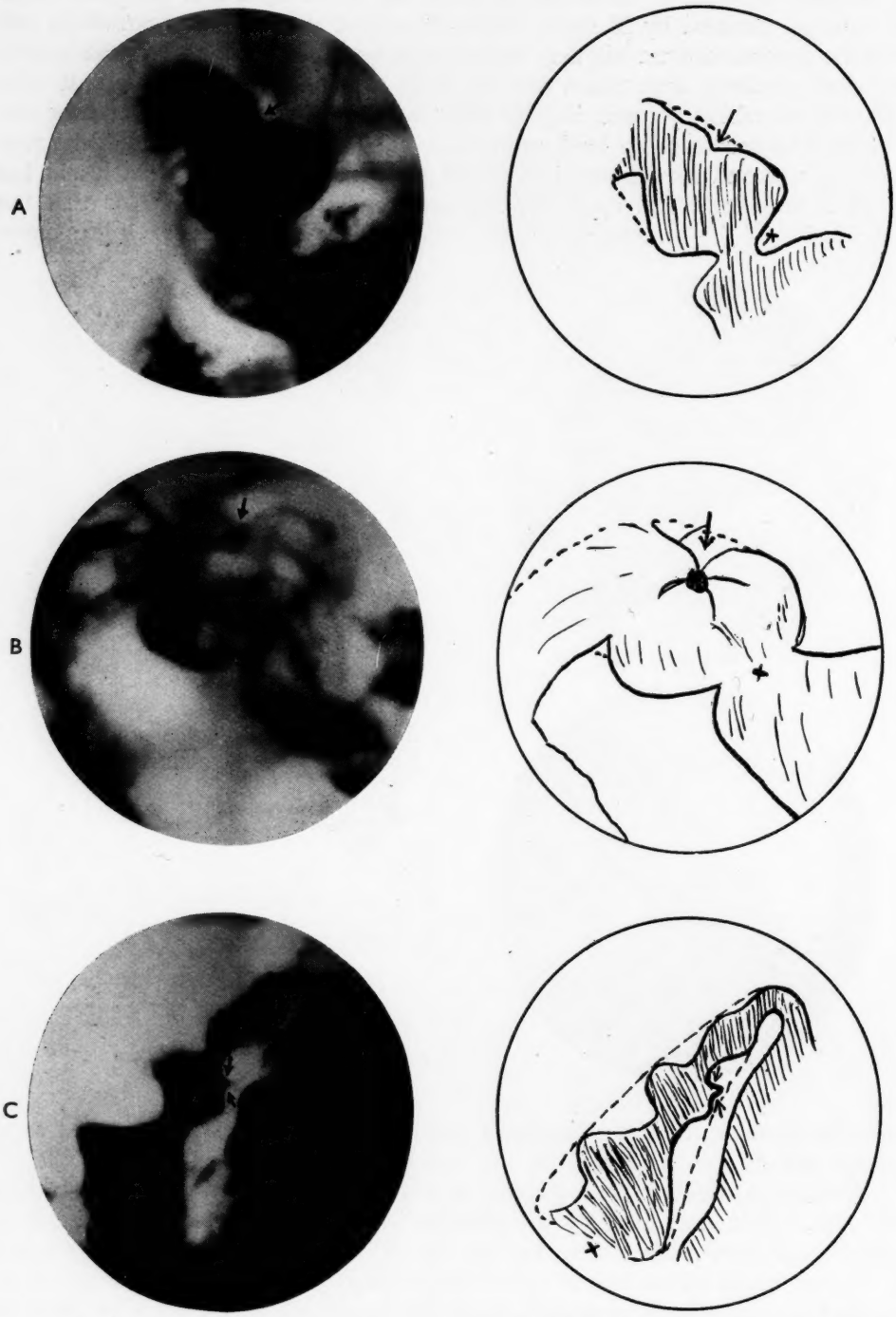

FIG. 488.—A, Aimed exposure. Circular narrowing of cap which is otherwise normal. B, Aimed exposure with dosed compression, showing *en face* niche with converging mucosal folds at site of narrowing. C, Niche shown in profile on posterior wall. Gross deformity of cap in this view is due to inflammatory swelling of the mucous membrane, the result of duodenitis. X, Pylorus.

FIG. 489.—A, Aimed exposure showing typical deformity of duodenal cap. B, Aimed exposure with dosed compression. Scarring changes of healed ulceration. No ulcer niche present. X, Pylorus.

FIG. 490.—A, Aimed exposure showing deformed cap. Cicatricial deformity of greater curvature. Inflammatory deformity of lesser curvature close to pylorus. B, Aimed exposure with dosed compression. *En face* view. Niche on anterior wall. Scarring changes on posterior wall with stiffened folds. C, Profile view. Niche localized on anterior wall. Posterior wall shortened, with loss of normal convexity, the result of scar-contraction of healed ulceration. X, Pylorus.

FIG. 491.—A, Aimed exposure. Typical deformed duodenal cap. B, Aimed exposure with dosed compression. *En face* niche in mass of scar tissue. C, Profile view. Niche on posterior wall. X, Pylorus. (Confirmed at operation.)

The deformities of the duodenal cap—the trefoil, the coral-like, the pine-tree, to mention but a few—are of importance. Yet a deformity of the cap, as the term is generally understood, is not the cardinal sign of ulcer, and should not be so used. Firstly, in more than 50 per cent of cases of duodenal ulcer the classical deformities are not present (*Figs.* 487, 488). Secondly, whilst it is true that a contour deformity of the cap may, and frequently does, signify the presence of an ulcer, this is not necessarily the case. As an ulcer involving the deeper layers of the duodenum heals, fibrosis and scarring occur. It is to this in the main that the classical deformities are due. It is natural, therefore, that deformities can be shown in patients whose ulcers have healed and who are free from symptoms (*Fig.* 489). Deformities of the cap are due or are ascribed to many conditions: duodenal ulcer, healed ulcer, duodenitis, cholecystitis, appendicitis, incomplete or fleeting filling, and periduodenal adhesions. The possibilities of misinterpretation are obvious and make deformity of the cap too unreliable a sign on which to base the diagnosis of duodenal ulcer. Moreover, if an ulcer is present, it is possible even in the most grossly deformed cap to isolate and demonstrate the ulcer niche (*Figs.* 490, 491). The importance of a deformity lies, not in its value as a sign diagnostic of ulcer, but as evidence of alteration in the lumen of the cap and thus a potential or active cause of gastric dysfunction.

The radiological picture corresponds to a plastic impression of the mucosal surface of the duodenal cap. The conditions prevailing there at the time of the radiological examination do not necessarily show on the serous side. This applies particularly to ulcers which are comparatively superficial, and have not penetrated to any extent into the deeper layers of the duodenal wall. Sometimes, therefore, after the radiological demonstration of an ulcer niche in the cap, the surgeon at operation is unable to detect evidence of ulceration. This is also true of some chronic ulcers, especially if they are situated on the posterior wall and at some distance from the pylorus where they are difficult of access. Further, if an interval has elapsed between the barium meal examination and the operation, the ulcer may have healed as the result of medical treatment. Before the radiological diagnosis is refuted, the duodenum must be opened and the inner aspect of the cap examined. Also every case in which a period of time has intervened should be re-examined a day or two before operation.

With the isolation and recognition of the ulcer niche the nature of the lesion is clear. No other radiological evidence is necessary for diagnosis. If no ulcer niche can be isolated, the statement that an ulcer is present in the duodenum at the time of examination is radiologically unjustified.

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ENCRUSTATION OF THE BLADDER AS A RESULT OF ALKALINE CYSTITIS

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ALTHOUGH calcareous deposits of a somewhat localized extent are not infrequently observed in the more chronic types of urinary disease, generalized encrustation of the bladder wall is far from common. The following case, occurring in a young patient, illustrates several characteristic features of the advanced condition.

CASE REPORT

M. P., a woman of 26 years who was the mother of one child and again in the early months of pregnancy, entered hospital on Jan. 29, 1935. Slight urinary frequency following a confinement three years previously was not attended by serious inconvenience. The onset of lower abdominal discomfort accompanied by extremely frequent and painful micturition—each of four days' duration—caused the patient to seek advice.

Systemic investigation disclosed no unusual findings, except that the offensive alkaline urine contained albumin, phosphates, and pus—a profuse growth of *B. coli* being subsequently obtained on culture. Radiological examination showed a clearly defined bladder outline (*Fig.* 492). A capacity of only 4 oz. and troublesome hæmorrhage rendered cystoscopy difficult, but after prolonged irrigation good views were secured. The irregularly contracted bladder walls were heavily coated with whitish encrustations (*Fig.* 493), bleeding necrotic areas being visible when fragments were detached. Besides the extensive surface deposits, stalactitiform masses were freely suspended, and sharply defined plaques occupied the vault (*Figs.* 494, 495). The ureteric orifices tended towards the golf-hole type, and specimens from each renal pelvis were acid although the bladder urine was strongly alkaline.

Numerous attempts were unsuccessful to isolate the *B. proteus ammoniæ* from the urine, and relevant tests failed to give any evidence of genito-urinary tuberculosis.

After twelve days' treatment with acid bladder irrigations (acid acetic B.P. 2 drachms to a pint of water) twice daily, cystoscopy showed a remarkable change. The interior was now free from all phosphatic concretions, but well-defined areas of ulceration were present in the mucosa (*Fig.* 496). Mercuric oxycyanide (1–4000) was substituted for the local acetic acid treatment. On viewing the bladder after ten days, recurrence was obvious on the anterior wall, the deposits now taking the form of smaller plaques surrounding the shallow ulcers. After re-instituting the acid lavage for another six days, further improvement ensued; the ulcers were smaller, of the healing type (*Fig.* 497), and only two small encrustations remained. Acid irrigations were continued for another six weeks.

When examined on May 10, 1935, after a fortnight without local treatment, the patient was free from urinary symptoms. The bladder capacity was 10 oz. and thin stellate scars occupied the sites of the former ulcers. No concretions were present, the left ureteric orifice was of normal appearance, and the right only slightly dilated. The patient was discharged on May 22.

After a normal confinement at the end of August, she returned for further observation (as requested) on Oct. 19. Cystoscopy revealed only the scarring of the previous ulceration without any trace of phosphatic deposits, although no treatment had been given since discharge.

DISCUSSION

Encrusted cystitis as defined by François[1] is a more or less localized ulcerative inflammation of the bladder wall with deposits of phosphate of lime on the surface and in the walls of the ulcerations.

After prolonged studies, Hager and Magath[2] came to the conclusion that encrusted cystitis with alkaline urine is caused by the implantation of *B. proteus ammoniæ* in a bladder which is already the seat of some form of inflammatory or

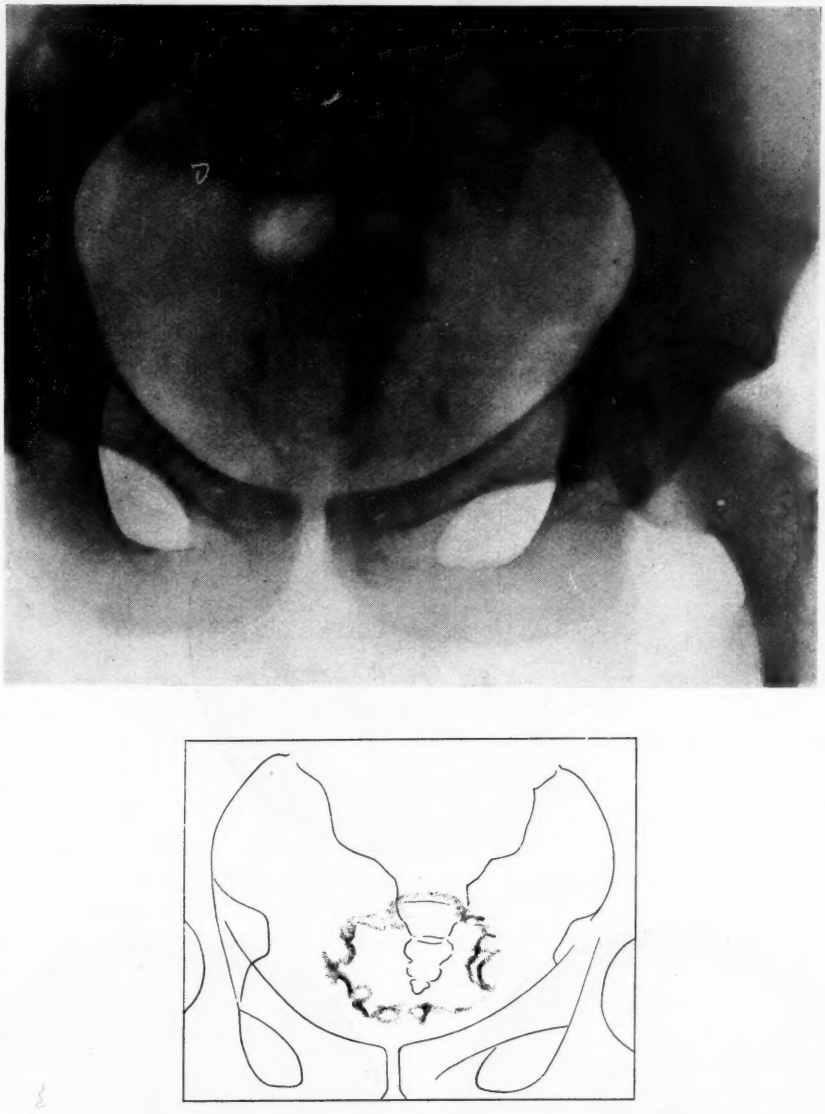

FIG. 492.—Skiagram showing crenated outline of encrusted vesical walls.

tumorous lesion. The encrustations result from the breaking up of urea into carbon dioxide and ammonia with the precipitation of alkaline inorganic salts.

The earlier writers on this subject observed the condition almost exclusively in women. A more recent review by Hager[3] of fifty cases at the Mayo Clinic shows the disease to be over twice as common in females as in males. He states that the condition prevails in married women and during the child-bearing period,

FIG. 493.—Cystoscopic view of encrustations. Bladder wall irregularly contracted as seen in skiagram. Mid-field simulates appearances of calculus. Soft downy deposits are seen projecting from the right.

FIG. 494.—Elevated or tumour-like encrustations of the stalactite type.

FIG. 495.—Flat or surface deposits, with sharply defined irregular edges, on the inflamed bladder wall.

FIG. 496.—Ulcer as observed twelve days after treatment began.

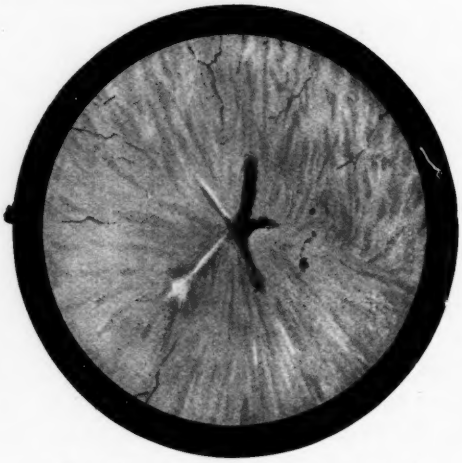

FIG. 497.—Healing ulcer—one month after treatment began.

the presumption being that trauma of the bladder and catheterization are contributing factors. In males, however, the disease appears more usually during the prostatic age.

The symptoms are those of severe chronic cystitis of variable duration—often preceded by attacks of a milder nature. Hæmaturia is repeatedly mentioned as a salient feature, and the passage of calculous material has been sometimes noted. The urine is generally alkaline, cloudy, and foul-smelling; it contains mucus, blood, and a varying quantity of pus. Specimens collected from the kidneys, however, are sometimes distinctly acid even when the bladder urine is alkaline—a fact frequently commented upon in urological literature.

Cystoscopy renders the diagnosis certain, but is often difficult, complicating factors being the high degree of vesical irritability and the tendency to profuse hæmorrhage. Some cystoscopical appearances may now be briefly mentioned.

1. *Cystitis* is as a rule very marked, particularly in the neighbourhood of the encrustations; œdema, which may obscure the ureteric orifices and even involve the urethra, is said to be a feature.

2. *Calcareous deposits* characterize the disease: these are firmly connected with the bladder wall and are of widespread or local distribution. According to the writings of Caulk[4] two varieties are encountered either alone or in association.

a. The flat or surface type—more commonly seen behind strictures, prostatic obstruction, around vesico-vaginal fistulæ, and capping bladder tumours.

b. The elevated or tumour-like type—this is the rarer and is due to constant precipitation of salts on an already encrusted ulcer.

3. *Ulceration*, which appears to be the essential lesion, occurs in any area of the bladder. Multiple lesions of varying size are commonly found, the condition in reality being one of chronic ulcerative cystitis. The irregular ulcers serve as nidi for precipitation of salts and usually involve the mucosa, but occasionally penetrate more deeply.

François was of the opinion that encrusted cystitis never disappeared spontaneously and did not readily yield to medical treatment.

Rendering the urine acid by the administration of such drugs as sodium acid phosphate and ammonium chloride is of little avail in ameliorating this infection. The beneficial effects of bladder lavage with acid solutions are usually very transient, the rapid disappearance of the concretions being followed, in the great majority of instances, by an equally rapid re-formation. More favourable results are expected from prolonged acidification by ketogenic diets or their more recent chemical substitutes. Caulk is an advocate of the intravesical injection of Bulgarian bacilli, the idea of such treatment being to have the organisms proliferate and produce an acidity which would be incompatible with the life of the organisms responsible for the disease.

Numerous surgical methods have been employed to eradicate the diseased mucous membrane, which forms the basis for the calcareous formations. Hager favoured intensive local treatment. Writing in 1926, he stated that experience has taught that the most consistently satisfactory results were obtained by the removal of all encrustations by means of curettement and topical applications of strong silver nitrate solution to the denuded areas.

In view of the well-known experiments on vitamin deficiency factors in urolithiasis and of the researches of Hunter[5] and others on calcium metabolism

attempts have been made to treat this form of cystitis on more general lines. Redewill[6] quotes an instructive recurrent case. By intensive vitamin therapy and the administration of parathyroid the tendency to recurrence was completely checked.

SUMMARY

Some noteworthy features of the present case may be recapitulated :—

1. The onset of the attack in the early months of pregnancy.
2. Almost the entire bladder was affected.
3. The bladder wall was outlined in skiagrams.
4. Although the voided urine was strongly alkaline, specimens secured by ureteric catheterization were acid.
5. Repeated examinations failed to isolate the *B. proteus ammoniæ* from the urine.
6. The remarkable benefit which followed lavage with acid solutions appears to be unusual.

We wish to express our thanks to Mr. Thornton Shiells for his illustrations of this article.

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SPONTANEOUS RUPTURE OF EXTENSOR POLLICIS LONGUS TENDON ASSOCIATED WITH COLLES'S FRACTURE

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THE object of this paper is to report three cases of spontaneous rupture of the extensor pollicis longus tendon associated with Colles's fracture.

CASE REPORTS

Case 1.—Miss R., aged 25 years, fell on her left wrist on Dec. 19, 1933. When seen the next day she was found to have a Colles's fracture with dorsal rotation of the lower fragment but no radial displacement. The fracture was reduced under local anæsthesia,

FIG. 498.—*Case* 1. The patient is unable to extend the interphalangeal joint of the thumb.

FIG. 499.—*Case* 1. Showing the result obtained by tendon transplantation.

a non-padded plaster cast applied, and active movements of the fingers and thumb encouraged from the beginning. Plaster was removed on Jan. 10, 1934. The fracture was healed in good position. The patient had 50 per cent of normal range of movement in the wrist and inferior radio-ulnar joints. Finger and thumb movements were practically normal. Physiotherapy (aerated baths and active movements) was started. On Jan. 21, 1934, the patient discovered that she could not extend the terminal joint of the thumb (*Fig.* 498).

Examination showed healed Colles's fracture with full function of all joints but inability to extend the interphalangeal joint of the thumb. She was unable to make the medial boundary of the anatomical snuff-box prominent. No movements of the interphalangeal joint of the thumb could be obtained by electrical stimulation of the extensor pollicis longus muscle. On Feb. 6 Mr. Gordon Irwin explored the tendon through a vertical incision 5 in. long across the dorsal aspect of the wrist. The tendons crossing the lower end of the radius were exposed, but that of the extensor pollicis longus was missing from its groove on the medial side of Lister's tubercle. Its place was taken by some yellowish soft tissue. The distal end of the tendon was found just below the lower edge of the dorsal carpal ligament. It was flattened, soft, and frayed. The proximal end was bound down to the bone just above the site of fracture and was much attenuated. End-to-end suture was not possible. The distal end was pulled through the tendon of the abductor pollicis longus and sutured to it with fine catgut sutures, the thumb being held in full abduction and extension. The wound was closed. The hand was put in splints which held the wrist in full dorsiflexion and the thumb in full extension and abduction. The splint and sutures were removed after three weeks, and physiotherapy started (aerated baths and movements). The patient was discharged on March 6 with a functionally normal hand (*Fig.* 499).

Case 2.—Mrs. B., aged 58 years, fell on her right arm on Dec. 11, 1933, and was seen in the fracture clinic the next day, when she was found to have a right Colles's fracture with no displacement. A non-padded plaster cast was applied and immediate active movements encouraged. The plaster was removed on Jan. 3, 1934. On Jan. 31 the patient complained of being unable to extend the interphalangeal joint of the thumb, and was found to have all the signs of rupture of the extensor pollicis longus tendon. The tendon was explored on Feb. 6 by Mr. Gordon Irwin. It was found in a condition similar to that of *Case* 1. The distal end of the tendon was transplanted into that of the abductor pollicis longus. The patient was discharged on June 4 with a functionally normal hand.

Case 3.—Mr. H., aged 57 years, slipped in the street on March 15, 1934, and was seen in the fracture clinic the same day, when he was found to have a right Colles's fracture with dorsal displacement and rotation of the lower fragment but very little radial displacement. It was reduced under nitrous oxide anæsthesia and a non-padded plaster cast was applied. The plaster was removed on April 5. On April 15 the patient noticed that he could not straighten the end of his thumb. Examination showed that he was suffering from rupture of the extensor pollicis longus tendon. I explored this on April 27, when the usual picture of spontaneous rupture of the tendon was found. The distal end was transplanted into the tendons of the abductor pollicis longus and the extensor pollicis brevis. The patient made a good recovery, except for a temporary slight median nerve paralysis due to the tourniquet which was used during the operation. He returned to work on July 9 with a functionally normal hand and reports he has been doing full work since.

DISCUSSION

Normal tendons are so strong that their rupture is rare. When great strain is placed upon them they usually tear away from their periosteal attachment, often with a flake of bone, as is commonly seen in the extensor tendons of the fingers, or solution occurs at the musculo-tendinous junction. Adams has reported a very interesting case of the latter type. A seaman jammed his hand in a doorway. Drawing the hand away he sustained a traumatic amputation of the distal phalanx of the middle finger, attached to which were 15 in. of the tendon of the flexor digitorum profundus which had torn away at the musculo-tendinous junction.

When a tendon is weakened by disease, rupture may occur during ordinary use, constituting the so-called 'spontaneous' rupture; 'pathological' rupture would be a better term. In cases of definite infective tenosynovitis such a result might be expected. Mason collected ten cases from Kanavel's records of tuberculous tenosynovitis.

Much more fascinating are those cases in which pathological tendon rupture is a consequence of trauma. The extensor pollicis longus tendon is classically the one affected. The condition was first described as affecting the hands of drummers in the German Army, giving rise to ' drummer's palsy '. The tendon, the seat of a chronic aseptic inflammation due to the repeated trauma sustained in the use of the left drum-stick, undergoes gradual solution. Later cases of rupture of this tendon were reported following injuries about the wrist, usually but not always associated with a fracture of the radius. In 1932 McMaster reported a case and could find only twenty-seven recorded cases in the literature dealing with the condition.

The etiology of the affection has been much disputed. It has been pointed out that the tendon with the wrist dorsiflexed makes two distinct angular bends—one round Lister's tubercle, and the other round the lower end of the dorsal carpal ligament. Any weakening of the tendon would lead to its giving way at one of these sites. Various writers have noted that the tendon in its osseo-fibrous tunnel has a very limited mesotendon. Weigeldt has shown that the vascular supply decreases after twenty-five years of age. Practically all reported cases have occurred after this age. Axhausen thought that the irregularity of its bony groove following the healing of a fracture might lead to the gradual fraying and solution of the tendon. Others believe that the damage occurs at the time of fracture, possibly caused by a sharp fragment of bone. Kleinschmidt produced a typical radius fracture in cadavers and showed that the tendon of the extensor pollicis longus was lacerated by a loose fragment of bone in every case. In Ashhurst's case the tendon had become adherent in the callus and this had apparently led to the rupture. In most of the recorded cases the condition had been attributed to tendon damage at the time of injury or mesotendon damage with consequent disturbance of the vascular supply.

The rupture usually occurs between seven days and seven years after the injury (Honigmann), although three weeks to three months is the average period. The rupture occurs without any pain or excessive use, but in one case it was preceded by a violent muscle cramp.

The three cases here reported occurred in the first five hundred patients treated in the Orthopædic Department of the Royal Victoria Infirmary, Newcastle-upon-Tyne, by the non-padded plaster cast with immediate active movements as advised by Böhler. When patients so treated are encouraged to move the part, it is often noted that finger movements are free and painless, while movement of the thumb occasions pain. As no case has been recorded in the hospital out of many hundreds treated previously with immobilization for seven to ten days followed by massage and movement a possible etiological factor is suggested. In a Colles's fracture the tendon of the extensor pollicis longus owing to its anatomical position must be contused. Active use might thus lead to its gradual solution at one of the sites of angulation. In one of the cases there was no displacement of the bony fragments so that no damage could have been inflicted upon the tendon by them. In another there was some comminution of the lower fragment, and a sharp spicule of bone is visible on the X-ray films, so that primary laceration of the tendon described by Kleinschmidt might have occurred. The intervals between the occurrence of the fracture and the spontaneous tendon rupture were 31, 33, and 51 days. At operation in each case the tendon ends were swollen, softened,

and rather yellowish in colour, the lower end being just distal to the dorsal carpal ligament, and the upper proximal to the site of fracture. There was no marked deformity of the lower end of the radius.

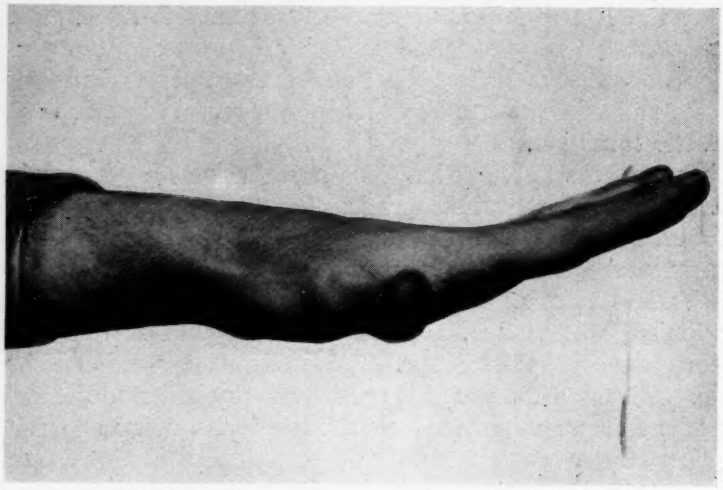

FIG. 500.—A normal hand: showing hyperextension of the thumb.

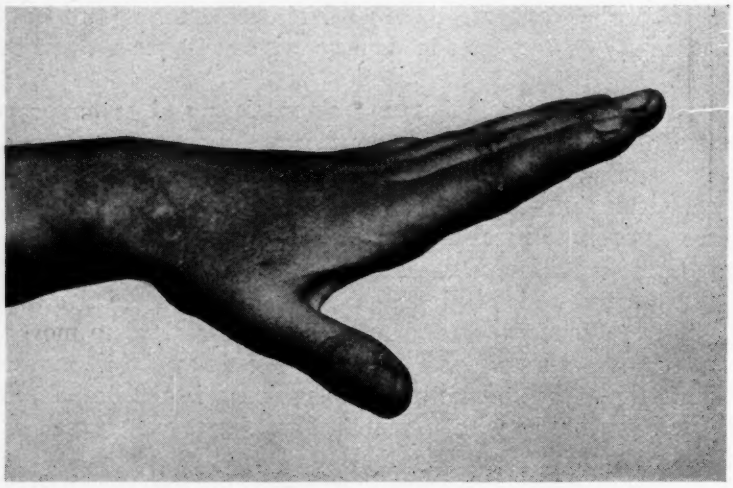

FIG. 501.—*Case* 1. Extension of the thumb. There is volar displacement of the thumb.

End-to-end suture was not possible, so in each case the distal end of the tendon was transplanted into that of the abductor pollicis longus. Good functional results were obtained, the patients saying that their hands were no different from what they had been before the accident. It was noticeable, however, that hyperextension of the thumb was not possible, and the extended thumb was slightly displaced volarwards as compared with normal (*Figs.* 500, 501). Another piece

of evidence of the loss of the normal extensor pollicis longus tendon was that it was impossible to place the thumb against the radial side of the index finger (*Figs.* 502, 503).

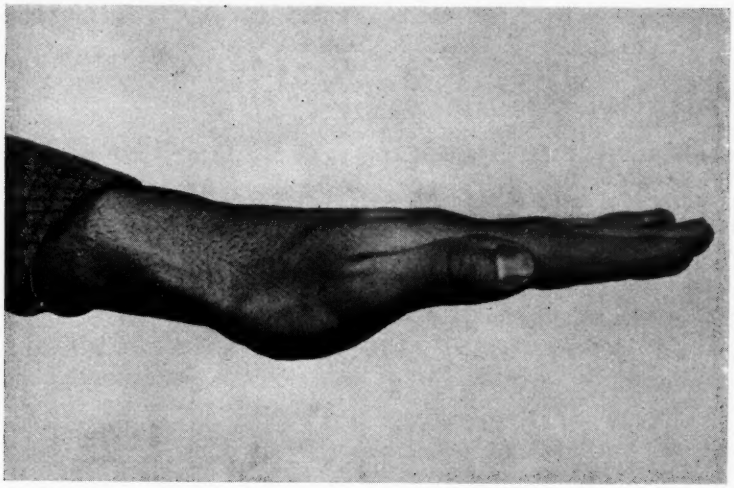

FIG. 502.—A normal hand: the thumb can be placed along the radial side of the index finger.

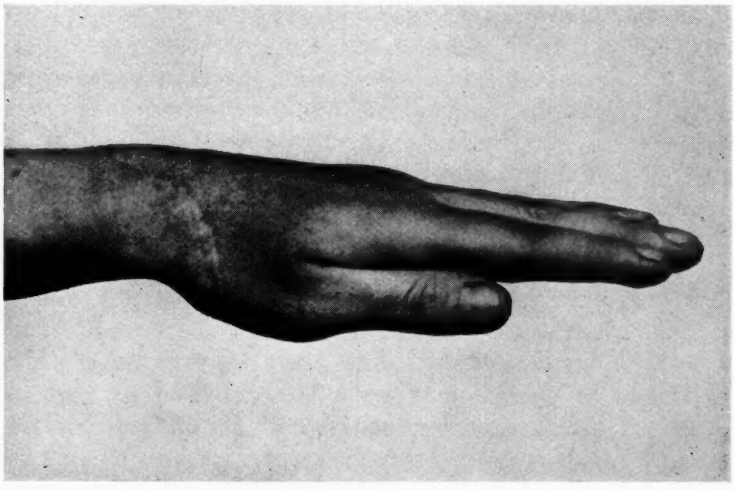

FIG. 503.—*Case* 1. The thumb cannot be placed along the radial side of the index finger.

SUMMARY

1. Three cases of spontaneous rupture of the extensor pollicis longus tendon associated with Colles's fracture are reported.

2. It is suggested that early use of the thumb has an etiological significance.

3. Good functional results were obtained following transplantation of the distal end into the abductor pollicis longus tendon.

4. The literature is reviewed.

My thanks are due to Mr. C. Gordon Irwin, under whose care the patients were, for permission to publish these cases and for his helpful advice and stimulating interest.

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SOLITARY PLASMOCYTOMA OF LONG BONES

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THIS condition, which shows considerable similarity to endosteal sarcoma, is of interest partly on account of its rarity but mainly because of the better prognosis it offers to life and limb than the far more common osteogenic growths.

Both the origin of the cells and the pathogenesis of the tumours are very debatable points. There are two main theories with regard to the histogenesis of the cells: (1) That they are derived from blood-forming elements; and (2) That they arise from the adventitial cells of the small blood-vessels. No final opinion can be given, but clinically the evidence is suggestive of the latter origin, for if the first were true, one would expect plasma cells to be a not uncommon occurrence in the blood instead of a rarity; also, though several cases of pseudo-leukæmia[12, 13] with associated involvement of lymphoid tissue are recorded, only one case of plasma-celled leukæmia has been reported.[14] Opinions are again divided on the equally vexed question whether the tumours are neoplastic or granulomatous in nature. The consensus of opinion of those who have studied the bone cases is that the condition is a true neoplasm, for, as Stewart points out, the plasma cell is the only cell present, it is very variable in size but tends to be larger than that found in any known granuloma, and mitotic figures are frequent.[8] Ewing agrees with this view,[15] but Kaufmann and others whose chief experience is with the extra-osseous lesions consider it may be a granuloma.[16]

Plasma-celled tumours are by no means confined to bone; in fact cases now have been reported from many regions of the body, and next to the multiple osseous form the most common situation appears to be the nasopharyngeal, including the buccal cavity[8] and larynx. Here the growths may be pedunculated, sessile, or infiltrative[17, 18] in character, most if not all are locally malignant, and a few give rise to glandular metastasis. Claiborn and Ferris[19] point out that the average age is 47 years, that 84 per cent are males, and that the presenting symptom in 75 per cent of cases is nasal obstruction, the rest complaining of epistaxis, dysphagia, hoarseness, or swelling in the neck. The prognosis is far better than that of sarcoma, for which it is usually mistaken, local excision giving satisfactory results in most cases, though there is on record one patient in whom multiple myelomatosis developed eight years after tonsillectomy for plasmoma.[20]

Other cases have been reported from the conjunctiva,[21] the pleura,[22] the bowel,[23] the skin,[24] and also as a cause of enlargement of multiple lymphatic glands.[25]

When arising in bone it, together with neoplasia of blood-forming cells and marrow endothelioma, forms the group known as 'myeloma', in which in days gone by osteoclastomata and even some osteogenic sarcomata were placed.

Geschickter and Copeland,[26] in their excellent analysis of 425 cases of multiple myeloma, point out that the plasma cell is the neoplastic element in about half

the cases of this disease. Elsewhere[27] these authors say, "Exceptions are so rare that the statement that myeloma is always multiple and always involves the trunk is practically vindicated." Though exceptions are distinctly rare, they are of interest and occur in the orbital region,[28] spine,[29] and long bones. Of the latter I have been able to find 12 cases in the literature since 1897, when Ewald[1] described the first case, and it is on an analysis of these and the case reported here for the first time that this paper is based.

CASE REPORT

T. S., male, coal miner, age 39. Under the care of Mr. J. B. Ferguson Wilson. Admitted July 26, 1935, to the Sheffield Royal Hospital with a fracture of the right tibia.

HISTORY.—Early in April, 1935, he knocked his right shin against a metal bar; it was painful for several days, but it did not stop his working. Three weeks later he noticed a swelling at the site of injury which has progressively increased in size since. On May 28 a fall of coal struck the swelling; he managed to work in spite of pain for three days, but then was obliged to give up for a week. After this he attempted to resume his employment but was forced to abandon it on the first day through pain at the site of the swelling. He continued to get about at home with the aid of a stick until July 12, when he fell over a mat and has been confined to bed ever since on account of pain when attempting to bear weight on the leg. The patient's previous history and family history are irrelevant.

ON EXAMINATION.—The patient was a healthy-looking man of strong build. There was a fracture at the junction of the upper and middle thirds of the right tibia in the centre of a soft swelling, which, however, only increased the diameter of the limb by about 2 in. There were no other positive findings.

Urine.—No Bence-Jones proteose or other abnormality.

Blood.—No abnormality in the blood-counts or morphology of the cells. The Wassermann reaction was negative.

X-ray.—Endosteal sarcoma with pathological fracture of the right tibia (*Figs.* 504, 505). No abnormality found in the skull, spine, thorax, pelvis, or upper parts of the femora.

Aug. 12.—Biopsy with diathermy.

Aug. 14.—Pathological report: Large round-celled sarcoma.

Aug. 15.—Amputation of leg through the lower third of the thigh some three hours after deep X-ray therapy.

Aug. 18.—The section was referred to Dr. H. E. Harding, who diagnosed the growth as a plasmocytoma. (*Figs.* 506, 507.)

Aug. 31.—Discharged.

December, 1935.—General and local conditions excellent.

DIAGNOSIS

Before considering special diagnostic points the following general clinical features may be noticed. The first symptom in every recorded case of solitary plasmocytoma of bone was pain localized to the affected area, though this cannot be of a severe nature as only 3 cases sought advice for it. From two to about six months after the onset of the pain pathological fracture occurred in 10 of the 13 cases, and 2 others developed such fracture while under observation. In none was there a marked swelling, and no case showed any of the systemic signs associated with multiple myelomatosis such as—Bence-Jones proteose in the urine, chronic nephritis with urea retention but low blood-pressure, leuco-erythroblastic anæmia, chronic pulmonary infection, or gastro-intestinal disturbance.

Average Age.—The average age of recorded cases is 48 years. In this the condition shows a marked difference from the other primary bone growths, for, as Ewing[30] states, "The incidence of osteogenic sarcoma reaches its acme at

the 15th year, thereafter rapidly declining; giant-celled tumour occurs most frequently in the 3rd decade; endothelial myeloma (or Ewing's sarcoma) is notable for its occurrence in early life, more than half the cases appearing before the 20th year."

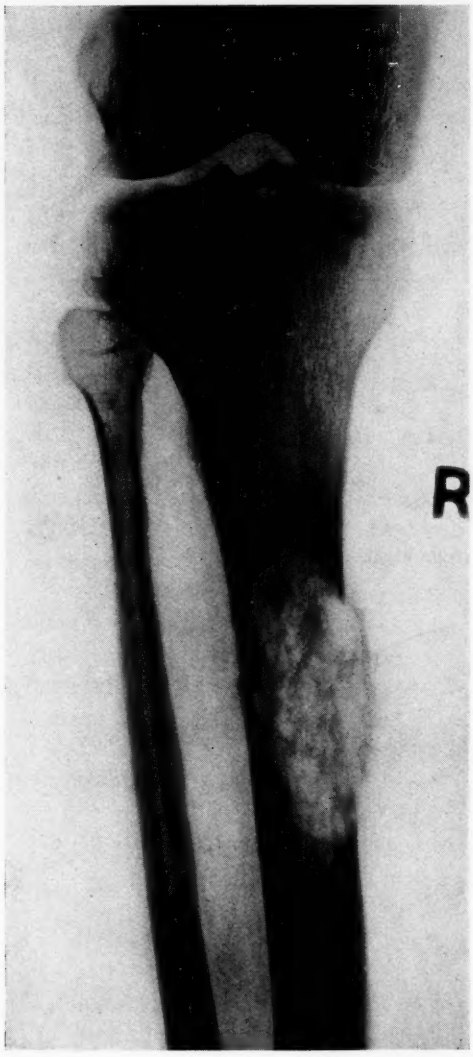

FIG. 504.—Antero-posterior view of plasmocytoma of the tibia.

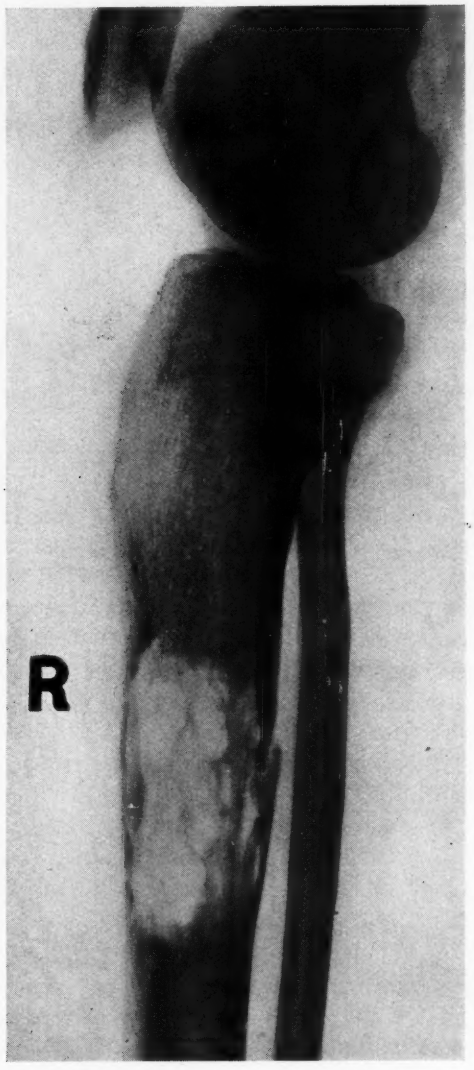

FIG. 505.—Lateral view of plasmocytoma of the tibia.

Sex.—Only 3 of the 13 cases were females, and this shows a greater proportion of males than osteogenic sarcoma, endothelial myeloma, and osteoclastoma, which are 3 : 2, 2 : 1, and equal respectively.[30] All the female cases were in the femur.

Bones Involved.—Femur, 8 cases; humerus, 3 cases; and 1 case each in the clavicle and the tibia.

Site.—Every case except one, which arose in the great trochanter and spread along the neck of the femur to the acetabulum, originated in the shaft of a long bone. In this respect plasmocytoma resembles an endothelioma, but the latter

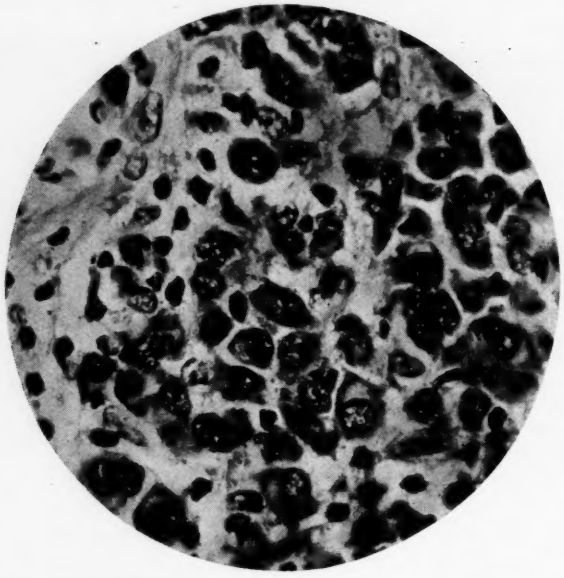

FIG. 506.—Microphotograph of section from plasmocytoma of the tibia.

FIG. 507.—Drawing to show cells stained by Pappenheim's method for plasma cells.

tends to present as a low-grade osteomyelitis and occurs in younger subjects. Osteogenic sarcoma and osteoclastoma occur practically invariably at the metaphysial and epiphysial regions respectively.

Pathological Fracture.—This occurred in 12 of the 13 cases, it was the reason for seeking medical attention in 10, and is the outstanding characteristic. It occurs frequently in telangiectatic bone sarcoma,[31] but the young age, rapid growth, early metastasis, and fatal termination make the diagnosis clear. Some 13 per cent of osteoclastoma show this condition, but the large swelling and the site of origin are distinctive.[32] Osteitis fibrosa cystica is commonly associated with pathological fracture, but the localized form is only found in youth.

Radiological Appearance.—This condition presents two distinct types of radiological appearance, both exceedingly suggestive. From those records in which it is possible to gather the appearance the commonest type is a solitary cyst in the shaft of a long bone. Extension takes place markedly in the long axis of the bone compared with the transverse, and neither periosteal nor osteogenic reaction is ever found. The second type shows a radiogram indistinguishable from that of an osteoclastoma, except that the site is different, which gives a clue to the diagnosis. Pathological fracture is almost invariably present in both. In the first group the age of the patient, and erosion rather than expansion, differentiate it from osteitis fibrosa cystica. Endothelial myeloma shows a periosteal reaction but no cyst formation, while endosteal sarcoma tends to present relatively less axial extension, greater lack of definition in the area of rarefication, and an osteogenic reaction may be present. In secondary carcinoma metastases from the thyroid and hypernephroma give rise to a shadow that is very similar, but the spread is more centrifugal; the diagnosis must be clinched by the clinical findings of the primary growth or noting the great vascularity or even expansile pulsation of the bone lesion.

Histological Appearance.—This is the only certain method of making the diagnosis, though it is of interest to record that with the exception of endothelial myeloma I have been unable to find any other type of solitary myeloma of long bone in the literature. Myelocytoma is occasionally found as a solitary lesion in the spine or skull, but erythroblastoma and lymphocytoma are apparently only found in the diffuse form of multiple myelomatosis. Sections show plasma cells of varying size with mitotic figures amongst them.

RÉSUMÉ OF TREATMENT AND PROGNOSIS IN RECORDED CASES

Deep X-ray Therapy.—This has been tried in three cases:—

1. Successful: a femur; the only case in the series without a pathological fracture; alive, well, and with no disability three years later.[6]

2. Failure: a humerus; radiation was combined with Coley's fluid; retrogression occurred at first, but as there was no tendency for the pathological fracture to heal amputation was resorted to with success.[6]

3. Failure: a femur, which four months before had been cauterized with the electric cautery and the cavity wiped out with 50 per cent zinc chloride and had had two pathological fractures since operation, was brought back for radiation and Coley's fluid. The report stated that he was "gradually loosing ground".[5] It should be noted in passing that plasma-celled tumours are not radio-sensitive in other situations, possibly because they are the cell associated so frequently with chronic inflammation.[32, 33]

Radium.—This was tried in one case,[4] but unfortunately sepsis supervened and amputation became necessary. On post-operative examination of the femur no trace of the growth could be found, its situation being replaced by fibrous tissue.

Conservative Surgery.—There were three cases in this group :—

1. Successful : a humerus treated by curettage and bone-graft from the tibia, the patient being alive, well, and with no disability one year later.[2]

2 and 3. Failure : the femur was involved in both cases and treated by curettage without graft.[5, 10] One is mentioned above as having post-operative radiation, and the other died of cirrhosis of the liver four months later without signs of local improvement.

Radical Surgery.—Amputation was performed in 6 cases, and 4 of the patients were reported as alive and well from a few months to eight years later. Successful cases were : humerus[6, 8] (2), femur[4] (1), and tibia (1). Two cases with involvement of the femur died of operative shock following disarticulation through the hip-joint.[3, 7]

Results.—Two patients died of operative shock, one of ? pneumonia,[1] one of perforated duodenal ulcer,[9] one of cirrhosis of the liver,[10] one of cerebral hæmorrhage.[11] Of the 7 remaining alive, 6 were reported well, but the seventh had signs of local recurrence.[5] In no case did multiple myelomatosis develop, though this has been recorded when the original lesion was in flat bones.

SUMMARY

1. A case of solitary plasmocytoma of the tibia is recorded for the first time.

2. The condition is discussed after analysis of the 12 recorded cases.

3. Every case of primary tumour of the shaft of a long bone should have a biopsy performed in case it is a myeloma, and if found to be of endothelial origin treated by deep X rays, and if a plasmocytoma by curettage and bone-graft.

In conclusion I wish to thank Mr. J. B. Ferguson Wilson not only for allowing me to report his case but also for help in writing this paper, and for the X-rays I am indebted to the kindness of Dr. J. L. Grout.

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THE BLADDER FUNCTION IN SPINAL INJURY*

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OPINION is by no means unanimous on the behaviour of the urinary bladder following disturbances of its innervation. It is, however, commonly believed that the bladder still exhibits a considerable activity even after its complete isolation from the central nervous system. On this view the importance of the peripheral nerve ganglia as 'centres' for micturition has been repeatedly emphasized.

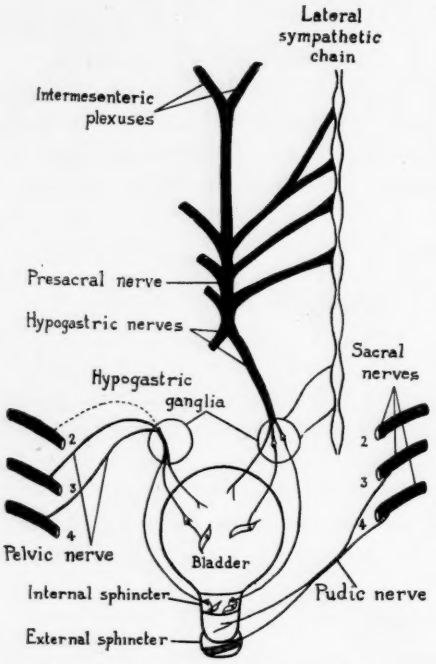

FIG. 508.—The innervation of the bladder. (*Modified from Professor J. R. Learmonth, 'Proc. Staff Meetings Mayo Clinic'*, 1931, vi, 182.)

This work was undertaken in an attempt to determine the exact function which exists in the bladder in cases of injury to the sacral segments of the spinal cord or cauda equina. More recently opportunity has occurred to study the behaviour of the bladder in cases of complete transverse lesions of the spinal cord. In these cases the influence on the bladder of the intact sacral segments of the spinal cord has been observed.

The Innervation of the Bladder.—The urinary bladder receives its nerve supply bilaterally from two sources (*Fig.* 508). These are the hypogastric nerves of the sympathetic nervous system, and the pelvic nerves (nervi erigentes) of the sacral parasympathetic outflow. The act of micturition is, however, importantly influenced by a third factor, the striated muscle of the sphincter mechanism, innervated by the internal pudic nerves.

The nerve-fibres of the sympathetic system proceed to the hypogastric nerves by various routes. In man the connector cells of origin are situated in the lower thoracic and upper lumbar segments of the spinal cord (about ninth thoracic to second lumbar). Their fibres pass to the lateral sympathetic chain, and from there the greater number reach the presacral nerve of Latarjet, which lies in front of the body of the fifth lumbar vertebra. This nerve divides at its lower end into the right and left hypogastric nerves. The hypogastric nerve passes on each side to the hypogastric ganglion, from which the fibres proceed to the pelvic viscera, including

* This work was first presented at a Hunterian Lecture at the Royal College of Surgeons of England on Jan. 31, 1934.

the wall and neck of the bladder. Some sympathetic fibres leave the sacral part of the lateral sympathetic chain and pass directly to the hypogastric nerve, and to the hypogastric ganglion (Latarjet and Bonnet,[6] Learmonth,[7] et al.).

The pelvic nerves (nervi erigentes) arise from the anterior divisions of the third and fourth sacral nerves. Latarjet and Bonnet[6] were not able to trace any connection with the second sacral nerves, but Learmonth[8] has evidence that the latter may sometimes play a part in the innervation of the bladder. Like the hypogastric nerves, the pelvic nerves also pass to the hypogastric ganglia.

The internal pudic nerves take origin from the second, third, and fourth sacral nerves, but, according to Rochet and Latarjet,[9] the striated fibres of the sphincter urethræ are suppplied chiefly by the third and fourth sacral elements.

It follows from this description that destruction of the third and fourth sacral segments alone will in most cases completely cut off the bladder from its innervation by the pelvic and pudic nerves, and leave it with its sympathetic (hypogastric) innervation alone.

Methods of Investigation.—Of the routine urological procedures no mention is necessary. It is essential, however, to refer to the technique of bladder pressure estimations, and to describe the findings in cases with a normal innervation. In this investigation a long vertical glass tube of about 4 mm. internal diameter was employed as the manometer. This was connected by a T piece with an inlying catheter and with a graduated reservoir for the introduction of fluid. The crest of the pubis, with the patient recumbent, was regarded as the 'zero' level. Two series of pressures were observed after each addition of 25 or 50 c.c. of fluid—those as the patient lay at rest, and again when he attempted to pass urine (Watkins[12]).

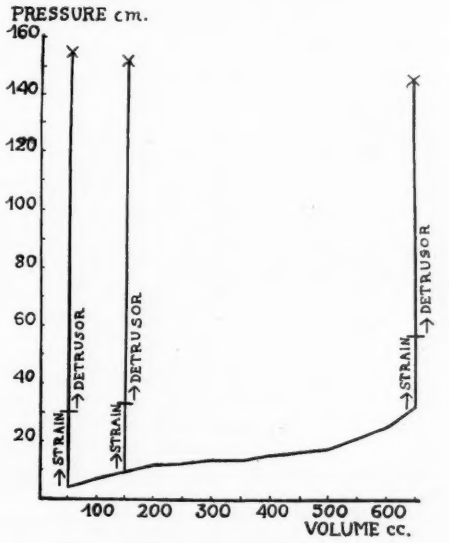

FIG. 509.—Bladder pressures in a case with normal innervation.

The characters of a bladder with normal innervation are shown in *Fig.* 509. The curve of the pressure as this man lay at rest shows a gradual rise as the volume within the bladder increases. His first sensation of anything within the bladder occurred with the introduction of 150 c.c. of fluid. On each occasion that he was asked to pass his urine the pressure increased rapidly by about 25 cm., and after an interval gradually and steadily to about 150 cm.; fluid was simultaneously voided around the catheter. The initial small rise of pressure is due to straining with the abdominal muscles, but the high pressure is the result of contraction of the detrusor muscle. Escape of fluid around the catheter is due to the associated reflex relaxation of the urethra.

Another example of normal innervation is shown in *Fig.* 510. When this man attempted to pass urine the resulting rise of pressure was usually only 3 to 5 cm., except at a capacity of 300 c.c., when reflex micturition was released, the pressure rising to 153 cm., and fluid being simultaneously passed around the catheter.

Recorded in the figures are also the pressures (47 and 34 cm.) which resulted when this patient was instructed to force as hard as possible with his abdominal muscles. These observations explain a type of pressure curve which is frequently found in bladders with normal innervation (*Fig.* 511). Here none of the earlier attempts to pass urine has released detrusor contraction, but the patient has increased the intravesical pressure to between 30 and 50 cm. by straining with his abdominal muscles as was apparent on watching his efforts. Only after the introduction of 600 c.c. did this man release a true detrusor contraction, the pressure rising to more than 150 cm.

Normal Micturition.—The two essential features of normal micturition are very clearly demonstrated by the above observations. Thus contraction of the detrusor muscle was shown to be very powerful, and promoted an intravesical

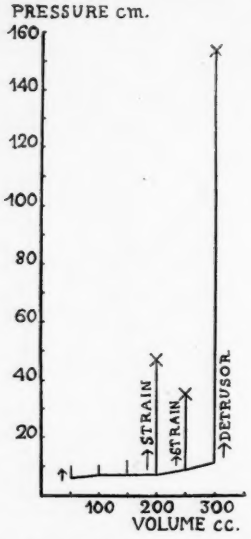

FIG. 510.—Bladder pressures in a case with normal innervation.

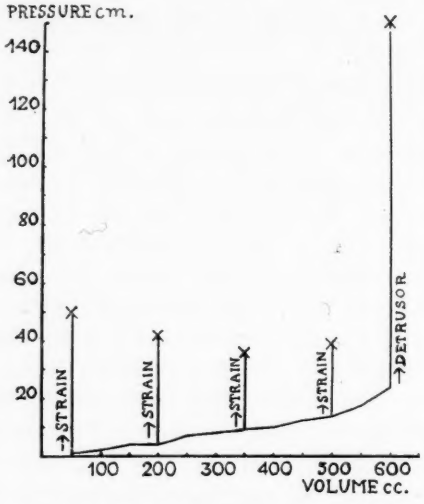

FIG. 511.—Bladder pressures in a case with normal innervation.

pressure much greater than that which could be achieved by straining with the abdominal muscles alone. The escape of fluid around the catheter revealed the associated relaxation of the sphincter muscles. It became very evident during observations on the normal bladder that abdominal straining plays no essential part in normal micturition. The difficulty which the normal individual may at times experience in releasing the micturition reflex was also very apparent. It proved that micturition could not be initiated in a manner comparable to the voluntary movement, for example, of a limb, and gave rise to the idea that voluntary micturition was dependent upon ' facilitation ' within the central nervous system. Denny-Brown and Graeme Robertson[3] have evidence that it is more accurately to be regarded as due to the removal of an inhibitory suppression of the micturition reflex.

CASE REPORTS

Case 1.—**Hæmatomyelia of the conus medullaris.**—

G. C., aged 19, was admitted to the Manchester Royal Infirmary on Feb. 24, 1928, under the care of Professor A. H. Burgess, who subsequently referred the case to Mr. Jefferson.

He had fallen a distance of 25 ft. from some scaffolding and had sustained a severe injury to his back. He was found to have a compression fracture of the first lumbar vertebra, with an injury to the conus medullaris.

Examination.—There was no paralysis or weakness of the muscles of the limbs. The knee-jerks were active and the ankle-jerks absent. There was a 'saddle' area of dense anæsthesia extending on to the posterior aspect of the scrotum. (This was more extensive than is indicated in *Fig.* 512, made at a later date.) Sphincter ani completely relaxed.

Bladder Function.—Retention of urine was absolute and was treated at first by intermittent catheterization and later with an inlying catheter.

Bowel Function.—No control.

On April 1, 1928, it was observed that the ankle-jerks had recovered, the left being more active than the right. One day during the later part of April (about two months after the injury) the inlying catheter became dislodged and the patient found to his surprise that he was able to pass some urine. He continued subsequently without recourse to catheterization.

On June 25, 1928, this patient attended as an out-patient.

Bladder Function.—There was no control at all during the night time, whilst during the day he could hold the urine for as long as two hours, though at times he might have to pass it every fifteen minutes.

On Oct. 5, 1931, he reported that he was working as a brick-building labourer.

Bladder Function.—He stated that the urinary stream was of very poor force, that it was frequently interrupted, and that sometimes it occurred only in drops. Incontinence at night time continued, and on most days he wet himself a little, always keeping a pad to soak up the urine which escaped. But he was quite certain that he could avoid incontinence completely during the day if he always went to urinate as soon as he felt the desire. A variable degree of frequency was still present, though often he would be able to hold his urine for three hours.

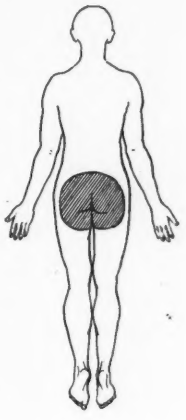

FIG. 512.
Case 1. Sept. 18, 1935.

Bowel Function.—There was marked constipation and frequent soiling of the clothes from the passing of flatus.

The Act of Urination.—The patient had not passed any urine for two hours and yet was unable to urinate on request. Firm pressure was made in the hypogastrium in an attempt to express urine. But although this was not successful it did promote the desire to urinate, and he then passed about 75 c.c. of urine in an interrupted stream. At first a few drops appeared, then a short stream, another few drops, another stream, and so on. The streams were of poor force.

Residual Urine.—This was found to be 300 c.c. immediately after the above act of urination. A considerable quantity of the urine was spontaneously evacuated through the catheter, but the latter part did not empty until after the application of pressure to the hypogastrium.

Cystography.—Sodium iodide (5 per cent) was introduced into the bladder, and skiagrams were taken immediately, and again after partial emptying of the bladder by catheter. This examination demonstrated a great degree of irregularity of the bladder and relaxation of the vesical orifice, the so-called 'funnel' urethra (*Fig.* 513). There is, however, no gross relaxation of the vesical orifice in the film made after partial emptying (*Fig.* 514). Regurgitation occurred up the right ureter and demonstrated a hydronephrosis and hydro-ureter on that side.

On Oct. 25, 1931, the patient felt so much improved that he had given up wearing pads to absorb urine.

Bladder Pressure Estimations.—After introducing a bicoudé catheter it was noticed as before that the last portion of the urine did not escape until assisted, on this occasion by straining efforts on the part of the patient, which resulted in the expulsion of another 50 c.c. or more of urine. The bladder-pressure curve (*Fig.* 515) exhibited some unusual features. The curve of 'rest' was an extremely steep one, and the first sensation of filling was noticed with only 75 c.c. of fluid in the bladder. The capacity, as indicated by the point of severe discomfort, was only 175 c.c., although the residual urine had previously been found to be

300 c.c. The pressures of 'attempted urination' were only 20 to 30 cm. greater than those of 'rest' and did not show any evidence of normal contraction of the detrusor.

On Jan. 23, 1933, he reported that he was in excellent health.

Bladder Function.—It was usually necessary to strain much harder than normally to urinate, though sometimes if desire was actually present the urine might come fairly easily.

FIG. 513.—*Case* 1. Cystogram (first film). Oct. 5, 1931.

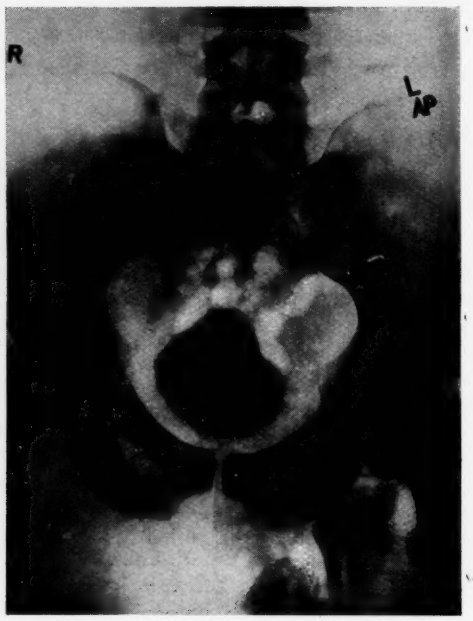

FIG. 514.—*Case* 1. Cystogram (second film). Oct. 5, 1931.

As a rule the urinary stream was interrupted many times, and there was marked terminal dribbling. The patient stated quite definitely that he could keep himself entirely dry by always responding quickly to the desire to urinate. However, he preferred to wear an apparatus to catch the urine whenever he left his home. Incontinence always occurred at night. He usually passed urine six or seven times during the day, and could hold it for as long as four hours at a time.

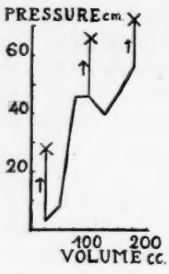

FIG. 515.—*Case* 1. Bladder pressures. Oct. 25, 1931.

Bowel Function.—Constipation was now less troublesome, but some incontinence still persisted; there was a tendency to soil a little from the passage of flatus or when attempting to urinate.

The Act of Urination.—On being requested to urinate this patient strained very hard, and contracted his abdominal muscles strongly. There resulted a stream of poor size and force, and this ceased instantly when the effort of straining was relaxed in order to take a deep breath.

Urine.—The urine was moderately cloudy and contained many leucocytes and some epithelial cells.

On Aug. 22, 1934, this patient reported again, complaining of abdominal pain, malaise, and more frequent urination with more troublesome incontinence of eight weeks' duration. Previously he had been doing splendidly. He would pass his urine six or seven times every day and would never get more than a very little wet in between times. Frequently he would wear an apparatus to catch what did come away, but at other times he would not trouble to use it even when going some considerable distance from home. At night time he was always incontinent. He stated that his urinary stream was interrupted. Examination revealed an abscess in the lower abdomen, which had apparently taken origin from one of the small diverticula of the bladder. After the abscess had been drained through a lower abdominal

incision urine leaked from the wound. Healing occurred with the help of an inlying catheter. During his stay in hospital (when he was bed-ridden) he had been persistently incontinent of both urine and fæces.

Cystoscopy.—The examination was made under ether anæsthesia with the concave sheath. There was a considerable amount of debris within the bladder. Trabeculation was considerable. The muscle bundles seemed to be fewer in number than is the case in obstructive conditions but they were very much more prominent and projected more markedly within the bladder cavity. Numerous orifices of diverticula were present. The ureteric orifices appeared normal. The vesical orifice was seen to be widely open, and one could examine the whole extent of the posterior urethra with the concave sheath.

On March 9, 1935, he had put on a stone in weight and was feeling fine.

Cystography.—A 15 Charrière rubber catheter was passed and readily entered the bladder. About 200 c.c. of 5 per cent sodium iodide was run into the bladder rapidly under a pressure of about 60 cm. This caused some discomfort, and a moment later the catheter slipped out of the bladder, followed by a little of the fluid. A skiagram was made when the fluid had ceased to run. A second film was made about thirty minutes later, at which time the patient was experiencing a good deal of smarting in the bladder. He then passed about 50 c.c. fluid, and another film was made three or four minutes later. The first two films showed wide relaxation of the internal sphincter and posterior urethra (*Fig.* 516), whilst in the third film no contrast medium could be seen in the posterior urethra (*Fig.* 517). The broad projection below the main bladder shadow seen in *Fig.* 517 is evidently a pouch from the bladder base. It can also be seen in the other films, in which it is partly superimposed upon the shadow of the dilated posterior urethra.

On Sept. 18, 1935 (seven and a half years after the accident), he reported himself as being very well.

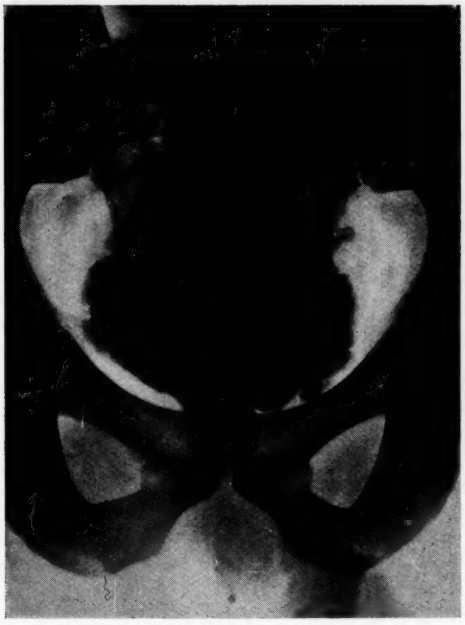

FIG. 516.—*Case* 1. Cystogram (first film). March 9, 1935.

FIG. 517.—*Case* 1. Cystogram (third film). March 9, 1935.

Bladder Function.—Sometimes he wears an apparatus and at other times just something in his trousers to soak up the urine. His urination varies greatly. He may occasionally be several hours without passing urine. At other times he may have to go three times within a few minutes. When he is incontinent he is certain that it is only small quantities at a time. As before, he can keep himself practically dry provided he is near a lavatory all

the time so that he can answer the call immediately. At night time he always keeps a bottle in bed and does not wake up.

Bowel Function.—Very constipated. He loses control if he has diarrhœa.

Examination.—Motor power excellent. Knee-jerks, active; ankle-jerks, left not quite as active as the right. There is a saddle area of diminished sensation to pinprick extending on to the back of the scrotum (*see Fig.* 512). Anal sphincter entirely patulous.

FIG. 518.—*Case* 1. Cystogram (second film). Sept. 18, 1935.

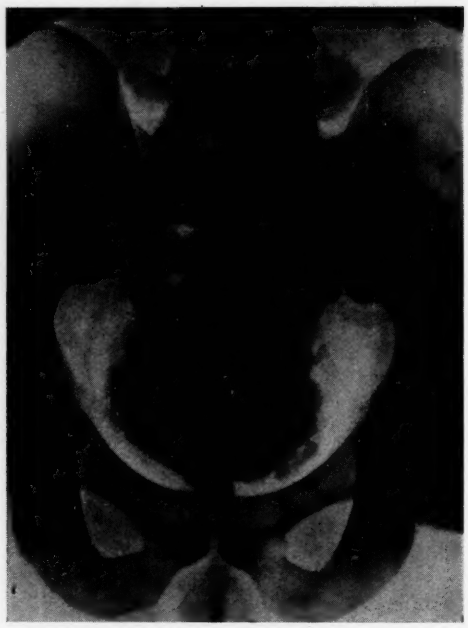

FIG. 519.—*Case* 1. Cystogram (fourth film). Sept. 18, 1935.

Combined Pressure Observations and Cystography.—He passed 100 c.c. of fairly clear urine. A rubber catheter (18 Charrière) entered the bladder readily and evacuated 170 c.c. of urine. Sodium iodide 5 per cent was introduced at a pressure of just over 80 cm. and the fluid was allowed to run in at full speed until he began to complain of the bladder being full, which occurred at a capacity of about 200 c.c. The inflow was stopped at 200 c.c. and the first X-ray was taken, the level in the manometer having fallen from that of introduction to a pressure of 60 cm. The pressure was then observed to fluctuate at intervals, e.g., from 40 to 65 cm., and then from 40 to 52 cm. The second film was taken when the pressure registered 38 cm. immediately after the pressure had fallen to this level following a contraction. Subsequent to this the pressure fluctuated from 38 cm. up to 65 cm. and down to 38 cm. again, and then up to 53 cm. In both these films (*Fig.* 518) there was the widest degree of relaxation of the vesical orifice and posterior urethra: 85 c.c. was then removed from the bladder, and the intravesical pressure registered 16 cm. A third film was taken at this pressure. After this the pressure was observed to fluctuate again from 16 cm. up to 35 cm. and back to 16 cm. up to 23 cm. and then back again. The fourth film was made when the patient again attempted to pass urine, and the pressure rose to 43 cm., the film being taken just as it had started to fall and registered 38 cm. In these two films there is relaxation of the vesical orifice and posterior urethra, but to a lesser degree than in the first films (*Fig.* 519).

Comment.—After a period of acute urinary retention following injury to the conus medullaris this patient developed a remarkable degree of control over his bladder function. The condition after seven and a half years shows that this was not due to recovery on the part of the innervation itself.

Case 2.—**Injury to the cauda equina.**—

W. C., aged 35, was admitted to the Salford Royal Hospital on Sept. 22, 1930, under the care of Mr. James B. Macalpine. Ten days previously, when he was working down a coal mine in a crouching attitude, he had been struck on the back by a heavy mass. He stated that his only complaint at first was that there seemed to be no sensation when his bladder was full, and that he only passed his urine once a day. However, two days before his admission to hospital, he felt a severe pain in his back when his nurse lifted him up in bed, and from that time urinary retention had been complete.

FIG. 520.
Case 2. Sept. 14, 1935.

Examination.—X-ray examination revealed a forward displacement of the fifth lumbar vertebra on the sacrum. All the muscles below the knees were weak with the exception of the right tibialis anticus. The left tibialis anticus acted less strongly than the right, but the long flexors and extensors of the ankles and toes did not show any action at all. All the other muscles were acting with the exception of the glutei. The knee-jerks were active and the ankle-jerks absent. There was an area of complete anæsthesia over the 'saddle' region, extending down the backs of both thighs and on to the penis and scrotum (*Fig.* 520). Sphincter ani entirely relaxed.

Bladder Function.—The complete retention of urine was at first treated by regular catheterization, but subsequently an inlying catheter was employed. A fairly bad cystitis developed at this time.

Bowel Function.—No control.

Cystogram (Oct. 3, 1930).—There was no evidence of a 'funnel' urethra or other abnormality at this time (*Fig.* 521).

On Nov. 14, 1930, some urine was voided for the first time, and three days later the residual urine was found to be 500 c.c.

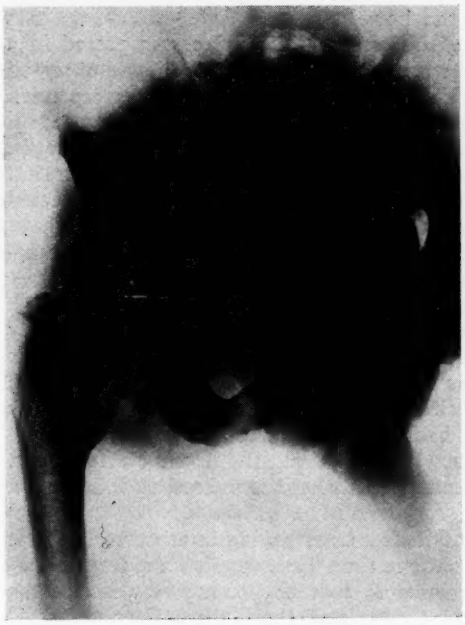

FIG. 521.—*Case* 2. Cystogram.
Oct. 3, 1930.

FIG. 522.—*Case* 2. Cystogram.
Nov. 10, 1931.

On Nov. 10, 1931, the patient reported for observation.

Bladder Function.—He stated that the urinary stream was only of moderate force. Occasionally slight incontinence of urine occurred, but he had not wet himself for the past two weeks. He could hold his urine for two or three hours at a time, but at night he kept a urinal in bed.

The Act of Urination.—He was observed to strain very hard, and was only successful after an interval. He then passed some urine in an interrupted stream, which was fine and of only moderate force.

Cystography.—This showed a very marked degree of irregularity of the bladder wall, and relaxation of the bladder neck or 'funnel' urethra (*Fig.* 522).

On Jan. 1, 1932, this patient again reported for observation.

Bladder Function.—He thought his urine was passing with less difficulty than before. He stated that it passed in a fairly good stream with some dribbling. With care there was no incontinence at all during the day, but if he neglected for long the desire to urinate a little would come away of itself. He still kept a urinal in bed. In the daytime he passed urine every hour or two but could sometimes hold it for three hours.

On Jan. 12, 1933, he stated that his general health was excellent.

Bladder Function.—This had improved very considerably. He thought that he did not have to strain quite so hard as before, but straining was a very definite factor, and, as he himself remarked, he never had to strain at all before his accident. Often he had to wait some time before the urine would come, and the stream was always interrupted several times and there was terminal dribbling. If he neglected to respond quickly to the sensation of bladder fullness, some incontinence would ensue; and if somebody made him laugh when his bladder was full, a few drops of urine would escape. But with care he was able to keep himself free from incontinence both by day and night. He passed urine five or six times every day, and always two or three times at night. The amount of urine would show much variation, being sometimes large but at other times quite small.

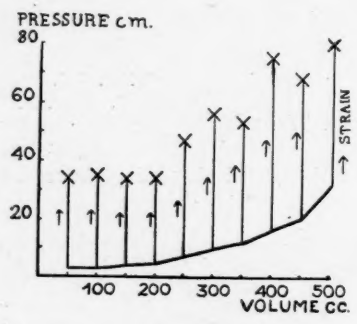

FIG. 523.—*Case* 2. Bladder pressures. Jan. 12, 1933.

Bowel Function.—He had not complete control of his bowels, and straining to urinate was liable to cause some fæces to pass. He also required frequent purgatives to work the bowels.

The Act of Urination.—The urine was passed in a series of small streams of fine calibre, and it was observed that the interruptions between the streams corresponded exactly with the times at which the patient paused in his straining efforts in order to take a deep breath. About 175 c.c. of urine was passed with about twelve interruptions of the stream.

Residual Urine.—This was found to be 200 c.c. The catheter was apparently not detected in the anterior urethra but it was felt as it approached the bladder neck.

Urine.—The urine was slightly cloudy and contained many leucocytes and Gram-positive cocci.

On Nov. 4, 1933, he again reported for observation.

Bladder Function.—He must always strain to pass his urine. If his bladder was very full, he said he did not have to strain very hard. And when his bladder was full he thought the urine came in a continuous stream, so long as he strained all the time. But otherwise the stream was always interrupted. Some terminal dribbling. Sometimes there was a very marked urgency, but at other times he could wait half an hour after the sensation first came on. He had not wet himself for several months now, either during the daytime or at night. Sometimes he passed urine every two hours, but often could hold it as long as four hours. Usually passed it twice every night.

Sexual.—Erections do occur, but not so strongly. Has been having intercourse but it is not satisfactory. Uncertain about ejaculations, but at one time urine would come from him.

The Act of Urination.—There was much straining so that he became very red in the face. The urinary stream was interrupted many times, and the interruptions occurred exactly whenever he took a deep breath. He passed 125 c.c., about 10 c.c. at a time, in a narrow stream of poor force.

Residual Urine.—This was 40 c.c., and it was necessary to express the urine by pressure applied to the hypogastrium.

Bladder Pressure Estimations (*Fig.* 523).—The first sensation from filling of the bladder was appreciated at 250 c.c. Each attempt at urination was associated with great effort of straining, and no evidence of powerful detrusor contraction was found.

Combined Pressure Observations and Cystography (Feb. 23, 1935).—Sodium iodide (5 per cent) was used. After passing a rubber catheter 150 c.c. of fluid was evacuated with the aid of pressure in the hypogastrium. Contrast medium was quickly run into the bladder and at 150 c.c. he noticed the sensation, and at 220 c.c. he experienced pain. The inflow was then closed and the first film was taken with the pressure at 28 cm. After an interval of some minutes the pressure had subsided to between 5 and 8 cm. at which point the second skiagram was made. The noteworthy point about these films (which are similar to those at a later date (*see Fig.* 524) is that they show a bladder of relatively smooth outline as compared with the cystogram of 1931 (*see Fig.* 522), and that there is no evidence of relaxation of the internal sphincter. It should be noted also that the bladder pressures did not rise higher than 28 cm. of water, in spite of the very rapid introduction of fluid.

Residual Urine (March 13, 1935).—230 c.c.

The man was next examined on Sept. 14, 1935, five years after the original injury.

Bladder Function.—He could now hold his urine as long as six hours in the night. He was still conscious of the necessity to strain to pass urine. He was not troubled by incontinence at all.

Bowel Function.—No control when bowels are loose. Constipated; taking purgatives twice a week.

Sexual.—He says his erections are improving, though not perfect. Intercourse is not very satisfactory on account of the anæsthesia, but he does get an orgasm.

Examination.—Movements of the right leg and foot good. Left leg wasted below the knee. Cannot move any of the toes of this foot. He can dorsiflex and plantarflex the foot moderately strongly.

Knee-jerks: Right active, left exaggerated. Both ankle-jerks absent. There is complete loss of sensation to pinprick over the 'saddle' area, backs of both thighs as far as the knee-joints, scrotum, and penis (*see Fig.* 520). No tonus can be detected in the sphincter ani.

FIG. 524.—*Case* 2. Cystogram. Sept. 14, 1935.

Act of Urination.—He was wanting to pass urine after being given a large quantity of water to drink. He now passed 500 c.c. urine. The first part came in a good strong stream of quite good calibre, but the latter half was very much interrupted, and there was some straining. But even with this part the force was fairly good, and the interruption was partly due to the manipulation of the penis with his fingers as a sort of 'milking' effect which seemed to help him to pass it.

Residual Urine.—150 c.c.

Combined Pressure Observation and Cystogram.—Sodium iodide 5 per cent was used. The fluid was introduced rapidly under a pressure of about 60 cm. The patient felt uncomfortable after the introduction of 250 c.c., and at 330 c.c. the inflow was stopped. The first X-ray was then taken, the pressure being 22 cm. water. After a minute or two the pressure was 11 cm. and a second X-ray was taken. He then attempted to pass urine, but only succeeded in raising the pressure to 40 or 50 cm., the third film being taken at the height of straining. In all these films the appearances were identical—smooth bladder outline without any evidence of weakness of the vesical orifice (even when the patient attempted to pass urine) (*Fig.* 524).

Comment.—The condition of the bladder in this case of injury to the sacral nerves was at first similar to that in *Case* 1. It is clear, however, that recovery of the bladder function had been taking place very gradually. Urinary incontinence

ceased to occur, and at the last observation urine was passed in a good strong stream, though emptying was still incomplete. The cystogram at the end of fourteen months showed gross irregularity of outline and marked relaxation of the vesical orifice; but these abnormal appearances were no longer present after five and a half years. The bladder pressure estimations were made three years after the injury, and it is probably also on account of commencing recovery that the curve was not as steep as might have been anticipated by comparison with *Cases* 1 and 5.

Case 3.—**Injury to the cauda equina.**—

W. McG., aged 7, was admitted to the Stockport Infirmary on April 7, 1930. He had been knocked down by a motor-car and sustained a fracture dislocation of the fifth lumbar vertebra, which was displaced backwards.

Examination.—There was no paralysis or weakness in the limbs. Knee-jerks active; ankle-jerks and plantar responses absent. There was loss of sensation extending over the 'saddle' area, down the backs of both thighs, and over the lateral parts of the legs and feet. (More extensive than is shown in *Fig.* 525, which represents a later date.)

FIG. 525.
Case 3. Nov. 21, 1933.

Bladder Function.—Retention of urine was present, and was treated by intermittent catheterization.

Bowel Function.—There was complete incontinence of fæces.

This patient was transferred to the Manchester Royal Infirmary under the care of Mr. G. Jefferson, and a laminectomy was performed on May 21, 1930. The dural sac showed a definite level of compression at the junction of the fourth and fifth lumbar vertebræ, and the arachnoid space was obliterated there. The nerve-roots were unruptured but rather gummy-looking below the compression. Recovery from the operative procedure was uneventful.

On July 20, 1931, considerable improvement was reported.

Bladder Function.—He could hold his urine for at least several minutes after the perception of the desire. He was passing urine voluntarily about six times a day, and did not get up at night.

Sphincter Ani.—This was extremely relaxed, and it was possible to expose the rectal mucosa by gentle traction on the skin around the anus.

The Act of Urination.—On being requested to pass urine this patient was observed to strain hard and contract his abdominal muscles. A stream of poor force was produced, which ceased when he stopped straining. With further straining the stream started again, and was interrupted several times in a similar way.

Suprapubic Expression.—This caused urine to escape in a stream which was continued only so long as the pressure was constantly maintained. The bladder could be palpated two inches above the symphysis pubis.

On Aug. 18, 1931, this boy was re-admitted to the Manchester Royal Infirmary for observation.

Bladder Function.—Following instrumentation urination became more difficult, and on a few occasions the bladder became very distended; the boy complained of severe pain, and was relieved by catheterization.

Cystography.—A large smooth bladder with a well-marked 'funnel' urethra was demonstrated (*Figs.* 526–528).

Bladder Pressure Estimation (*Fig.* 529).—The curve 'at rest' was not abnormal in type. The first sensation was of 'pain in the stomach' at 300 c.c. The pressures of attempted urination were about 100 cm., and showed the features characteristic of abdominal straining.

On Jan. 1, 1932, further progress was reported.

Bladder Function.—He occasionally wet his shirt very slightly, and rarely also he wet his bed. He passed urine every two hours or so, and once in the night.

The Act of Urination.—He was observed to pass about 50 c.c. of urine in an interrupted stream of poor force. Subsequently the bladder was palpable at a level about two inches below the umbilicus.

FIG. 526.—*Case* 3. Cystogram (first film). Sept. 7, 1931.

FIG. 527.—*Case* 3. Cystogram (second film). Sept. 7, 1931.

FIG. 528.—*Case* 3. Cystogram (third film). Sept. 7, 1931.

Suprapubic Expression.—Expelled urine in a stronger stream than was attained in urination.

On Jan. 12, 1933, the boy reported for observation and was in excellent health.

Bladder Function.—He stated that the urinary stream was usually interrupted. During the day he occasionally wet his shirt a very little, whilst at night he might go two or three weeks without trouble; last week, however, he was incontinent on two occasions. He passed urine four or five times during the day, and once or twice every night.

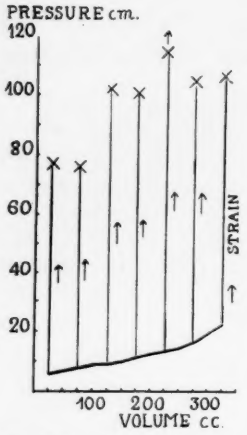

FIG. 529.—*Case* 3. Bladder pressures. Aug., 1931.

The Act of Urination.—He was seen to pass 150 c.c. of urine in a stream which was interrupted about ten or twelve times whenever he ceased straining to draw a deep breath. (The bladder had been palpable two finger-breadths below the umbilicus.)

Suprapubic Expression.—Caused urine to escape only so long as the pressure was continued.

On Nov. 21, 1933, three and a half years after the injury, this boy was again examined.

Bladder Function.—On most days he wet himself a little, and sometimes quite a lot if he stayed out too long playing with other children. The frequency varied considerably, but he could hold his urine for as long as six hours.

Bowel Function.—Frequently his clothes were soiled after taking purgatives, but he did not have any accident as long as his motions were solid.

Examination.—Contour and movements of lower limbs normal. Knee-jerks both active; ankle-jerks absent. Loss of sensation over the 'saddle' area and down the backs of both thighs to the level of the knees (*see Fig.* 525). The anus admitted two fingers, and no tonus could be detected. The fundus of the bladder was palpable mid-way between the symphysis pubis and umbilicus.

The Act of Urination.—It was evident that he expelled his urine by abdominal straining. He passed 175 c.c. with seven or eight interruptions. Subsequently 25 c.c. was expelled by pressure applied to the hypogastrium.

Urine.—Centrifuged deposit contained occasional epithelial cells only.

Comment.—The type of urination in this case of injury to the cauda equina was similar to that of the other cases in that it was entirely dependent upon abdominal straining. Some important differences in the behaviour of the bladder, especially the absence of resistance to filling, will be discussed below.

Case 4.—**Spina bifida with involvement of the sacral nerves.**—

R. L., aged 10, was admitted to the Chirurgische Universitätsklinik, Freiburg im Breisgau, Germany, on Feb. 20, 1933. He had apparently been quite healthy until two and a half years before, since when he had developed bladder and rectal trouble. The condition was found to be due to spina bifida.

Bladder Function.—He passed urine about every two hours. Often there was a slight wetting of the clothes, but this was never very much. He was always incontinent at night. He always knew when he wanted to pass urine.

Bowel Function.—He had no control over his bowels.

Examination.—The boy walked perfectly. The contour of the lower limbs was normal. There was a pressure sore over the right gluteal region. Limb movements were normal. Both knee-jerks were active; the ankle-jerk was present on the left side, but absent on the right. There was a 'saddle' area of complete loss of sensation to pinprick extending on to perineum, scrotum, and penis. This also extended down the posterior aspect of the right thigh (*Fig.* 530). The anal sphincter was entirely relaxed, admitting three fingers. Rectum full of fæces.

FIG. 530.—*Case* 4. March, 1933

The Act of Urination.—This was associated with much straining, and about 200 c.c. of urine was passed in a narrow stream of poor force, which was interrupted each time that the taking of a deep breath interfered with the straining. The abdominal muscles could be felt to contract during straining.

Suprapubic Expression.—Pressed out urine fairly readily, but the flow ceased instantly on releasing the pressure.

Urine.—Contained a few leucocytes.

Cystoscopy.—Under light intravenous anæsthesia cystoscopy showed the mucous membrane of normal appearance, but the bladder neck was widely open so that the whole posterior urethra as far as the colliculus seminalis was readily seen.

Comment.—This case also demonstrates the typical behaviour of the bladder in lesions of the conus or cauda equina. The characteristic nature of the urinary stream and its dependence upon abdominal straining were very clearly demonstrated.

Case 5.—**Spina bifida with involvement of the sacral nerves.**—

W. N., aged 7, first attended the Brady Urological Institute at the Johns Hopkins Hospital on April 6, 1932, and was subsequently under observation there for several months. When six weeks old he had been operated upon for meningocele and spina bifida, and at the age of three months for hydrocephalus. The child had never been able to walk on account of extensive paralysis in the lower limbs, and incontinence of urine and fæces had been present since birth.

FIG. 531.
Case 5. April 6, 1932.

Bladder Function.—The mother stated that the urine escaped by a more or less continuous dribbling, and that it never seemed to come in an actual stream except occasionally when the boy laughed. There was no control whatsoever.

Bowel Function.—No control.

Examination.—The child was of poor intelligence. There was a small cranial defect in the parietal region, and an operative scar in the lower lumbar region. A deep pressure ulcer was present in each gluteal region. All the muscles below the knees, the hamstrings, and the glutei were paralysed on both sides. There was bilateral congenital dislocation of the hips, with contractures of hips and knees, and bilateral drop-foot. Both knee-jerks were active; ankle-jerks absent. There was loss of sensation over the 'saddle' area, and the lateral surfaces of the legs and feet. Pinprick was appreciated to some extent over the posterior aspect of both thighs (*Fig.* 531). No anal sphincter tone could be detected. The rectal mucous membrane was readily exposed by means of very gentle traction on the skin.

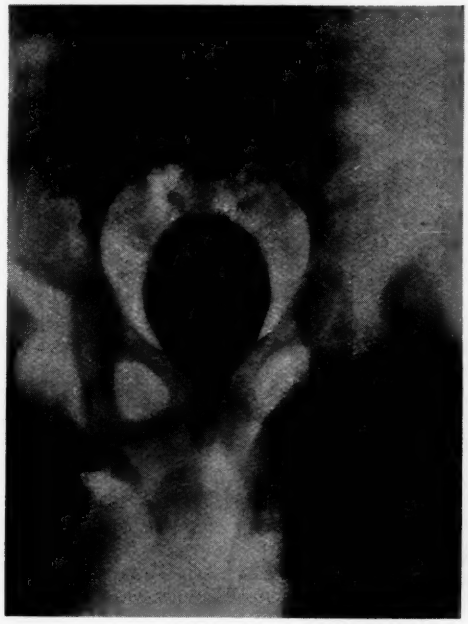

FIG. 532.—*Case* 5. Cystogram.
April, 1932.

Suprapubic Expression.—Urine (40 c.c.) was expressed without difficulty, the stream ceasing instantly on removing the pressure: 35 c.c. of urine was then found in the bladder. No leucocytes were to be found in the centrifuged urine.

Cystography (*Fig.* 532).—Showed a small ovoid bladder, without gross trabeculation. The vesical orifice was relaxed, the so-called 'funnel' urethra; there was a filling defect in the lower part of the latter which suggested an enlarged verumontanum.

Bladder Pressure Estimation (*Fig.* 533).—A 14F coudé catheter was employed. The curve was remarkably steep, so that a pressure of 37 cm. of water was recorded at a volume of 150 c.c. This degree of distension gave rise to discomfort, but not to actual pain.

Cystoscopy.—This was undertaken with a 'convex' sheath. The bladder wall appeared normal and without trabeculation. The vesical orifice was relaxed and its posterior lip was prominent. The verumontanum appeared pedunculated and mobile, and was almost transparent in appearance. On either side of the posterior wall of the urethra there radiated from the base of the verumontanum three ridges, of which the lowest were directed transversely outwards, the uppermost obliquely upwards and outwards. Appearing like congenital valves of the posterior urethra, they were, however, not sufficiently prominent to give rise to obstruction.

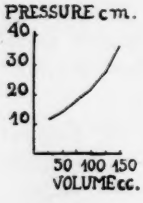

FIG. 533.
Case 5. Bladder pressures. April, 1932.

On two occasions urethral instrumentation was followed by urinary retention. Catheterization relieved this condition, 250 c.c. being evacuated the first time, and 150 c.c. the second time.

The mother was instructed to make the child strain at regular intervals in the hope that some degree of voluntary control might be effected. On July 18, 1932, the boy was observed whilst he strained to urinate, and a stream of poor force with two interruptions was noted. On Aug. 1, 1932, his mother stated that he could keep himself fairly dry if he strained at hourly intervals, but that he was completely incontinent at night.

Comment.—This boy of 7 years had had an extensive lesion of the cauda equina since birth. Up to the time at which he first came under observation the bladder condition was apparently one of more or less continuous dribbling. He was taught to gain some degree of control by straining with his abdominal muscles.

Case 6.—**Complete transverse spinal lesion.**—

M. H., a girl of 13 years, was admitted to the Manchester Royal Infirmary on July 9, 1932, because of pain in the back and loss of power in the legs. She became totally paralysed shortly after admission, with incontinence of urine and fæces. There was dense anæsthesia and complete paralysis below the eighth thoracic segment. An exploratory laminectomy was performed by Mr. Jefferson and Mr. Platt on Aug. 6, 1932, and revealed a sarcoma of the vertebral column. X-ray therapy was subsequently employed. The patient was examined on Nov. 13, 1933. The general health had remained good but complete paralysis and absolute anæsthesia had persisted below the level of the lesion (*Fig.* 534). There were deep pressure sores on the back.

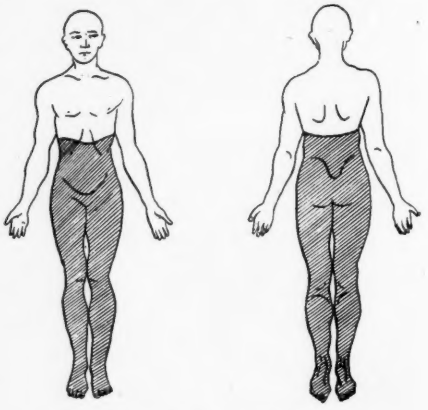

FIG. 534.—*Case* 6. Nov. 13, 1933.

The mother states that the child had absolutely no control whatsoever over urination, nor had she any knowledge of when the bladder was functioning. The mother did not know how often urination occurred, but she had observed the urine to come away in a strong stream and uninterrupted. She seemed to think that urination was sometimes brought on by movements. There was no control over the bowels.

Bladder Pressure Estimations.—A 14 Charrière rubber catheter was introduced and strapped to the thigh. There was rather more than 50 c.c. of cloudy urine in the bladder. Estimations of the intravesical pressure were made after the introduction of each 50 c.c. of fluid. The volume of fluid which had been introduced did not always indicate the volume within the bladder since some fluid was evacuated alongside the catheter and only 200 c.c. of fluid was found in the bladder at the end of the examination (400 c.c having been introduced). After each introduction of 50 c.c. the pressure was watched over a period of several minutes.

Volume Introduced	Pressure Changes
A few c.c.	After closing the inlet the pressure diminished slowly and at varying rates to 12 cm.
50 c.c.	Similar slow fluctuating fall to 29 cm., then a slight rise to 34 cm., falling to 23 cm., rising to 27 cm., and then falling to 12 cm.
100 c.c.	Fluctuating rates of fall to 19 cm., then to 16 cm., rising at a moderate and steady rate to 64 cm., falling in the usual fluctuating manner to 18 cm., and continuing again in a similar manner to 19 cm., to 17 cm., to 64 cm. by a steady rise, to 19 cm., to 43 cm., to 42 cm., to 60 cm.
150 c.c.	Fluctuating fall to 17 cm., steady rise to 68 cm., to 57 cm., to 69 cm., fluctuating fall to 24 cm., steady rise to 70 cm., to 38 cm., a small wave of contraction to 40 cm. interrupting the fall, which then continued to 17 cm. (*Fig.* 535).
200 c.c.	Fall to 16 cm., then up to 70 cm., to 59 cm., to 74 cm., to 24 cm.
300 c.c.	57 cm., to 69 cm., to 59 cm., to 66 cm., to 15 cm.
350 c.c.	42 cm., to 53 cm., to 42 cm., to 52 cm., to 36 cm., to 50 cm., to 17 cm.
400 c.c.	Fall to 18 cm.

The considerable rises of pressure which occurred always took place at a steady and fairly rapid rate. The fall of pressure always occurred much more slowly, at varying rates, and with short interruptions. Sometimes a small wave of increased pressure would interrupt the gradual fall. The pressure variations over a period of several minutes following the introduction of 150 c.c. fluid into the bladder are recorded in *Fig.* 535.

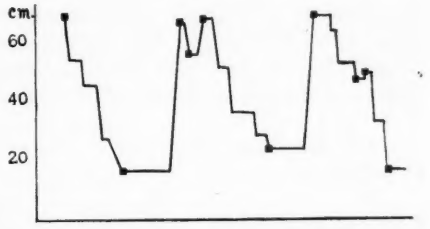

Fig. 535.—*Case* 6. Diagram of the pressure changes occurring spontaneously over a period of a few minutes. (Exact time not noted—about five minutes.) Nov. 13, 1933.

Comment.—Quite powerful and apparently spontaneous contractions of the detrusor muscle were occurring at intervals in this case of complete transverse spinal lesion at the eighth thoracic segment.

Case 7.—**Complete transverse spinal injury.**—

G. D. N., aged 23, was admitted to the Manchester Royal Infirmary on Nov. 21, 1933, under the care of Mr. Wilson Hey and of Mr. Geoffrey Jefferson. He had sustained an accident four weeks previously when he fell a distance of 40 ft. whilst rock climbing. He was found to have a compression fracture of the first lumbar vertebra with paralysis of the lower limbs.

Bladder Function.—He stated that he did not pass any urine after the accident until he was catheterized at the end of forty-eight hours. He did not get any pain from the over-distension of the bladder and he felt no sensation on catheterization. Intermittent catheterization was first employed, and subsequently an inlying catheter. On the fifteenth day, when the catheter became dislodged, urine seemed to come away from him easily on having his bowels well moved. From this time onwards he was not catheterized, and the urine seemed to run from him all the time.

Bowel Function.—For the first two weeks after the accident the bowels moved only with enemata. Since then they have moved once a day with fair regularity, but he has no control.

On abdominal examination (Nov. 28, 1933) the bladder felt like a ball extending two finger-breadths above the symphysis pubis. The application of pressure caused urine to be evacuated in a stream. On releasing the pressure the stream ceased, but a little dribbling followed for a moment or two. It was recorded at the time that the flow did not stop as immediately as is usual in cases of injury to the sacral segments. On Jan. 7, 1934, expression of the bladder showed just the same features, pressure causing a stream of urine, a little dribbling persisting after the removal of the pressure.

Examination.—The condition as observed on Aug. 12, 1934, is recorded here. At this time, almost ten months from the time of his accident, the level of sensory loss was practically the same as on admission. He was not able voluntarily to move toes, ankle, or knee on either side. He could flex at the hip-joint on the right side, but only quite weakly on the left side. Knee- and ankle-jerks were absent on both sides. Stimulation of the sole on the right side caused an upward movement of the toes with dorsiflexion and slight abduction of the ankle, and an upward movement of the toes on the opposite side. Stimulation of the toes on the left side caused an upward movement of the toes and dorsiflexion of the ankle on the same side.

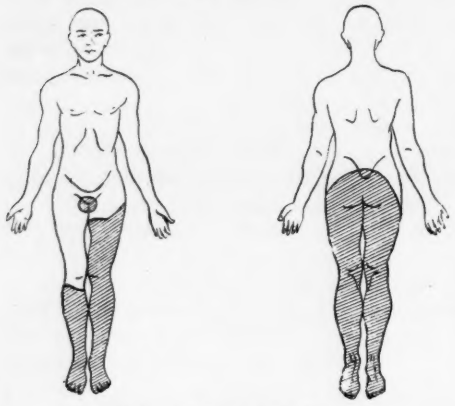

FIG. 536.—*Case* 7. Aug. 12, 1934.

There was dense anæsthesia over the buttocks, backs of the thighs, legs, and feet (*Fig.* 536). The anæsthesia also extended over scrotum and penis.

Sphincter Ani.—One finger was passed and there was felt to be excellent tone (100 per cent). When a second finger was gradually introduced the tonus persisted at first and then very suddenly gave way, leaving the anus entirely patulous. On removing the fingers the mucous membrane of the rectum was now visible through a wide orifice. Approach of the finger to the anal canal produced a strong contraction of the anal sphincter immediately it made contact with the skin at the anus. (An active condition of the anal sphincter had previously been noted on April 5, 1934, at which time it was thought that movement of the finger within the anal canal promoted rhythmical contractions, which at their height seemed to be 100 per cent.)

Bladder Function.—On Feb. 4, 1934, the passage of urine was watched over a period of two hours.

6.25 p.m.	Passed 55 c.c. in a continuous stream of moderate force and calibre, followed by a little terminal dribbling.
6.32 p.m.	He felt the sensation as if urine was passing, but no urine actually passed.
6.42 p.m.	Copious drinking of water commenced.
7.00 p.m.	Between 6.25 p.m. and 7 p.m. only one drop of urine had passed, and that just at the beginning of this period. He now felt the sensation which he associated with urination, and about ten seconds later passed 95 c.c. of urine in quite a good stream, with one interruption and a little terminal dribbling.
7.16 p.m.	A much stronger sensation occurred and about five seconds later he passed 85 c.c. in a quite strong stream with one interruption, followed by much terminal dribbling for about 15 seconds. When the penis was lifted to move the receiver a short spurt of urine occurred.
7.33 p.m.	Very quickly after the occurrence of the sensation he passed 130 c.c. in a good stream with one interruption.
7.36 p.m.	No response to stimulation of the penis with a pin.
7.42 p.m.	The sensation occurred followed quickly by the passage of 130 c.c. in a strong stream with one interruption.
7.43 p.m.	Attempted expression was unsuccessful. Quickly repeated sharp pressures in the hypogastrium were also without effect.
7.47 p.m.	Quickly repeated sharp pressures in the hypogastrium were followed by the sensation and then by the passage of 35 c.c. of urine in a stream of less than usual force. There was a short spurt, then a rather long interruption, and then another stream with one more interruption.

7.53 p.m.	Stimulation of the glans penis with a pin. After a moderately long latent interval 70 c.c. were passed in the usual way.
7.58 p.m.	Glans penis stimulated again. After 45 seconds he felt a weak sensation which recurred again after 60 seconds, but no urine escaped.
8.01 p.m.	The sensation occurred and five seconds later 125 c.c. passed with one interruption.
8.06 p.m.	Glans penis swabbed with methylated spirit. A small quantity of urine passed in small spurt.
8.07 p.m.	Swabbing glans penis was without effect.
8.10 p.m.	Glans penis swabbed again. After ten seconds he said, "It's coming", and in another ten seconds there passed 110 c.c. in a strong stream.
8.17 p.m.	Bicoudé catheter passed. 140 c.c. urine evacuated from the bladder with the assistance of some pressure to the hypogastrium.

The urinary stream seen during the course of these observations seemed of normal force and calibre. The single interruption lasted one or two seconds and occurred when about half the urine had passed. The stream then came on again with its original force which became gradually less and finally passed into a little terminal dribbling.

Bladder Pressure Estimation.—On Feb. 13, 1934, a bladder pressure observation was made. On catheterization only a few cubic centimetres of urine were found in the bladder, and this was only evacuated with the help of pressure applied to the hypogastrium.

Volume Introduced	Pressure Changes
A few c.c.	5 cm.
50 c.c.	The pressure fell to 30 cm. on closing the inlet, and then increased again gradually and in stages to 70 cm., and remained for a time fluctuating in this region, reaching a maximum of 85 cm. There was no associated straining.
60 c.c.	Pressure increased a little after closure of the inlet, and then gradually fell to 20 cm., during which time the patient complained of "aching in the kidneys".
100 c.c.	After closing the inlet the pressure only fell a little to 72 cm., and then increased to 96 cm., fluid escaping simultaneously around the catheter.

This patient developed a severe urinary infection, and at one time it appeared likely that he would die from pyelonephritis. X-ray examinations revealed stones in both kidneys and on occasion small stones were passed. An excretory urogram showed dilatation of the pelvis and ureter on both sides. The latter examination also showed a small bladder of regular outline, but it did not provide definite evidence of the condition of the vesical orifice.

Comment.—In this case there was recovery of reflex activity in the segments distal to the crush. Observation of the urinary evacuations, which were involuntary, revealed an active condition of the bladder. The urine passed in a stream whose force and calibre were normal. The presence of powerful detrusor contractions was also confirmed by observations on the bladder pressure. There had, therefore, developed a high-grade reflex, though involuntary, micturition. There was evidence that micturition could be set up reflexly by certain forms of stimulation.

Case 8.—**Hæmatomyelia, with subsequent recovery of bladder function.**—R. R., aged 50, was admitted to the Manchester Royal Infirmary on Aug. 13, 1931, under the care of Mr. C. Roberts. He had fallen a distance of 12 ft. and sustained a compression fracture of the first lumbar vertebra, with an injury to the lower spinal cord.

Examination.—On both sides all the muscles below the knee-joints were paralysed. The quadriceps extensor and hamstring muscles were weak but not paralysed. Flexion, adduction, and abduction of both hips could be performed. Knee-jerks and ankle-jerks

were absent. Both lower limbs showed an extensive dense anæsthesia, the anterior and lateral surfaces of the thighs alone remaining unaffected, as shown in *Fig.* 537. The anus was without tone, and two fingers were readily introduced.

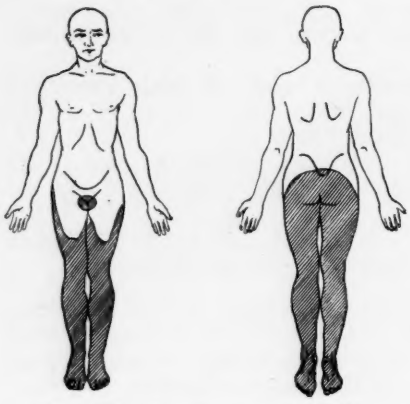

FIG. 537.—*Case* 8. Aug. 14, 1931.

Bladder Function.—Urinary retention was complete, and was treated by an inlying catheter.

Bowel Function.—There was no control.

Cystoscopy.—This was undertaken forty-eight hours after the accident. There was no trabeculation. No evidence of weakness of the bladder neck was observed, and the posterior urethra could not be brought into view with a ' concave ' sheath.

Cystography (*Fig.* 538).—Cystograms were made within twenty-four hours of the accident and on the 28th, 64th, 85th, and 132nd days. No evidence of weakness of the vesical neck could be found at any examination, even when unsuccessful efforts to urinate were made.

Bladder Pressure Examinations (*Fig.* 539).—During the early stage these observations were made on the 7th, 17th, 27th, and 54th days. All these curves were essentially similar. The capacity as indicated by severe discomfort varied between 550 and 650 c.c. Each curve of 'rest' showed a very gentle rise to a pressure of about 20 cm. of water at the full capacity. The curves of ' attempted urination ' were always low ; the greatest pressure attained was 87 cm. of water, but usually the pressure recorded was considerably less than this. The attempts to urinate were associated with very much straining, and the pressures could only be maintained momentarily. The first sensation of bladder filling was appreciated at these examinations with respective fillings of 400, 350, 250, and 200 c.c. The outstanding abnormal features at each examination were diminished appreciation of bladder filling and the absence of any sign of strong detrusor contraction.

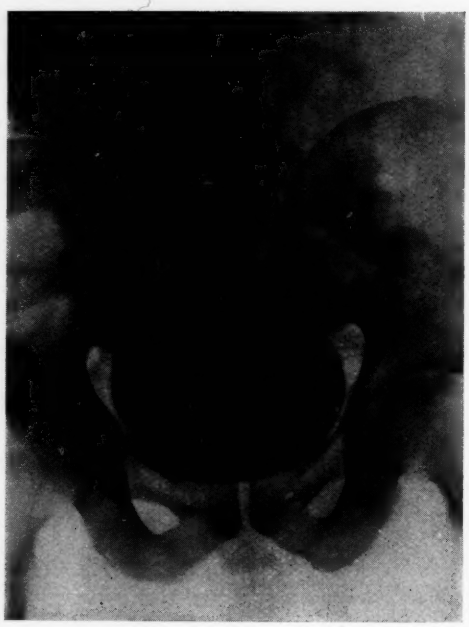

FIG. 538.—*Case* 8. Cystogram. Sept. 11, 1931.

Urine was first voided spontaneously on the 61st day, attempts on previous occasions having been without success. Occasional catheterization continued to be employed until the 75th day. Subsequently the passage of urine, which was at first a matter of extreme difficulty and associated with great effort, began to take place with increasing facility.

Residual Urine.—This was 550 c.c. on the 75th day. Subsequently it was estimated on a number of occasions to be rather more than 500 c.c. On the 116th day the residual urine was 300 c.c.

On the 116th day a bladder pressure estimation was made and certain remarkable differences from the previous examinations were evident. Pain was noticed after only 100 c.c. of fluid had been introduced into the bladder, and when 150 c.c. had entered the intravesical pressure overflowed the manometer (indicating a pressure of over 100 cm. of water) and urine escaped from the urethra alongside the catheter. These observations indicated improved bladder sensation, active detrusor contraction, and active relaxation of the urethra, and they demonstrated a returning function in the paralysed nerve-supply of the bladder.

A more complete series of observations were made on the 132nd and 133rd days.

Bladder Function.—The patient stated that he now usually passed urine every two hours during the day. When the desire to urinate was appreciated he could not wait for more than a very short time or it would come of itself. At night he wakened two or three times, and the urine came so rapidly then that he would wet the bed if he did not keep a urinal at hand.

Bowel Function.—This was now improved so that with suitable purgation the bowels moved once a day without incontinence.

Examination.—There was still almost complete paralysis below the knee-joints, excepting for some action of the left tibialis anticus. The hamstrings and glutei were very weak. The knee- and ankle-jerks were absent on both sides. The anal reflex was well marked from stimulation on either side. Pinprick was now appreciated as sharp all over the 'saddle' area. The posterior part of the right thigh was almost normal, but there was relative analgesia over the posterior part of the left thigh. Pinprick was definitely appreciated on the penis and scrotum. Gross anæsthesia to pinprick was present in both legs and feet up to the knee-joint on the left side, but not quite so high on the right side. The anal sphincter now showed definite tonus.

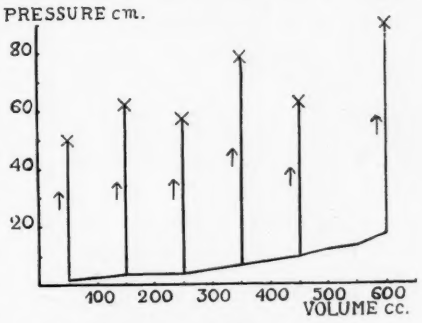

FIG. 539.—*Case* 8. Bladder pressures. Aug. 30, 1931.

The Act of Urination.—Whilst cleansing the glans penis preliminary to catheterization, urine was passed in a stream of considerable force with short interruptions. This action was quite involuntary. On the next day a similar phenomenon was observed when the penis was being examined by pinprick for analgesia. A sudden desire to urinate was aroused which passed off only after the passage of urine in an interrupted stream. After an interval of one minute the penis was again stimulated with a pin and after a few seconds desire to urinate was again manifested, and the interrupted stream was repeated. The abdominal muscles contracted strongly during the procedure but the whole process was apparently quite involuntary.

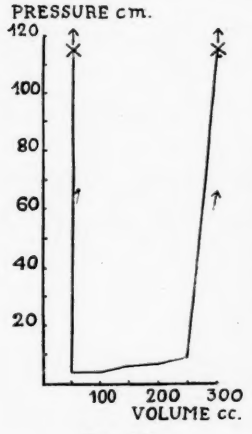

FIG. 540.
Case 8. Bladder pressures. Dec. 22, 1931.

Residual Urine.—On consecutive days this was found to be 300 c.c. and 175 c.c. respectively. All except the last few cubic centimetres was evacuated through the catheter in a strong stream.

Bladder Pressure Estimation.—In two consecutive examinations the curve of 'rest' was seen to be low and within normal limits. At the first examination, with 200 c.c. of fluid in the bladder, a sudden increase of intravesical pressure occurred so that the manometer overflowed (a pressure of more than 115 cm. of water). Bladder filling was appreciated with only 50 c.c. of fluid in the bladder. The result of 'attempted urination' was a pressure of over 115 cm. of water, even when there was only 20 c.c. of fluid in the bladder. At the second examination (*Fig.* 540) as the bladder filling was being increased from 250 to 300 c.c. a sudden rise of pressure occurred to over 115 cm. of water, and fluid was forcible expelled alongside the catheter. These observations confirmed those of the 116th day, demonstrating improved bladder sensation, active detrusor contraction, and active relaxation of the urethra.

The patient was next seen on Jan. 9, 1933, seventeen months after his accident. He reported a gradual steady improvement in his condition.

Bladder Function.—He stated that he did not have any further trouble whatsoever with bladder function. He did not now have to strain to urinate, and there was not any hesitation. He had not been troubled with incontinence at all since September, but when he first returned home this was a marked feature. The urinary stream was of good force and was not interrupted. Urine was usually passed five or six times a day, and he did not have to get up at night unless he took a large quantity of fluids in the evening.

Bowel Function.—He had complete control, and the bowels were moved regularly twice a day.

Sexual Function.—Occasional erections and nocturnal emissions occurred, and he thought that he would be able to perform sexual intercourse normally.

Examination.—He was quite unable to stand upright without support. There was marked wasting of the lower limbs. Abduction and extension of the hip-joints were weak. Quadriceps action was of only moderate power. Semimembranosus and semitendinosus were acting strongly, but no action of the biceps could be detected on either side. Practically no movements of ankles or toes could be undertaken except for a weak action of the left tibialis anticus. Knee-jerks were absent. Some ankle-clonus on both sides. Pressure over the anterior tibial muscles resulted in dorsiflexion of the toes on either side. Normal sensation was now present over the ' saddle ' area, the thighs, and the upper parts of the legs. There was still impairment of sensation in both feet and in the lower part of the left leg.

Comment.—Mr. Jefferson states that this must have been a scattered lesion of the spinal cord, with maximum damage to the fifth lumbar and first and second sacral segments.

It is very instructive to notice the different phases of bladder activity which were manifested in this case. The initial state was that of acute urinary retention. This passed on to a stage in which urination was evidently achieved by straining with the abdominal muscles which forced urine from a paralysed bladder. The extreme degree of effort associated with urination testified to that fact. Increasing facility of urination gradually developed, and the urinary stream regained a very considerable force. The evidence of this and of the bladder pressure observations showed that reflex micturition—powerful detrusor contractions associated with sphincter relaxation—had become re-established. For some long time, however, urinary control was absent, and the condition was one of involuntary reflex micturition as observed in the cases of transverse cord lesions, of which this was an incomplete example. Further evidence of the nature of the bladder function at that period is provided by the reflex facilitation of involuntary micturition which followed stimulation of the glans penis. At a still later time normal bladder function with perfect control became re-established.

DISCUSSION

BEHAVIOUR OF THE DETRUSOR MUSCLE

1. Lesions of Conus and Cauda Equina.—In striking contrast with the easy facility of normal urination, bladder emptying in these cases was a most laborious process. The efforts of straining were very considerable, and it was evident that the patient was utilizing his abdominal muscles to force out urine from the bladder. The urinary stream was interrupted at frequent intervals which always coincided with the moment at which the patient desisted in his straining efforts in order to take a deep breath. There can be no doubt that abdominal straining had replaced detrusor contraction as the expulsive force in urination. Furthermore, in no case did the observations on intravesical pressure reveal the powerful detrusor contraction characteristic of normal micturition. It can only be concluded that the detrusor muscle has lost its function of powerful contraction.

A surprising feature of these cases was the relatively small amount of fluid which could be injected into the bladder through a catheter. In *Case* 1 severe discomfort was produced by the introduction of 175 c.c. of fluid at a pressure of more than 60 cm., although there had been 300 c.c. of residual urine. This phenomenon was also observed in *Cases* 2 and 5.

The very steep curves of intravesical pressure, especially well marked in *Cases* 1 and 5 of this series, are manifestations of this same behaviour (*see Figs.* 515, 533). Both Schwarz[10] and Adler[1] were familiar with this type of curve in lesions of the conus and cauda equina. The explanation must be that the detrusor muscle reacts to rapid filling by a contraction which prevents distension reaching as great a degree as when, in the ordinary course of events, the bladder slowly fills with urine from the upper urinary tract. That actual muscular contraction must have taken place as the result of filling is demonstrated by the fact that the pressure subsides gradually if the volume is allowed to remain constant. The observations of Holmes[5] demonstrated this point very clearly.

Denny-Brown and Graeme Robertson[3] have shown that a contraction of the detrusor muscle may also be initiated by a momentary pressure applied to the bladder through the abdominal wall. Not only so, but these workers also recorded the occurrence of spontaneous waves of vesical pressure. These were waves of relatively small amplitude, producing pressure changes of not more than 20 to 25 cm. of water. Such spontaneous waves were observed only in *Case* 1 of this series, in whom well-marked spontaneous contractions were seen.

The bladder may not apparently exhibit this active and irritable condition in every case. In *Case* 3 the pressure curve did not rise steeply with increasing volume (*see Fig.* 529), and this, as well as the large size of the cystogram made after rapid filling with contrast medium (*see Fig.* 526), reveals no evidence of the resistance to filling so characteristic of the other cases.

2. Complete Transverse Cord Lesions.—A very different behaviour of the detrusor muscle was manifest in these cases, following recovery of reflex activity in the distal part of the spinal cord. Observations of the bladder pressure in *Case* 6 revealed a very active condition of that muscle. Sudden and considerable rises of pressure took place at intervals without apparent cause (*see Fig.* 535). This same phenomenon was observed by Holmes.[5]

In *Case* 7 observations on the intravesical pressure revealed similar powerful contractions of the detrusor muscle. These were also demonstrated very effectively by the powerful stream with which the urine was expelled from the bladder. The urinary stream was as forcible and as broad as that of normal micturition, and, quite apart from the fact that the patient was lying at rest without any straining, could only have been produced by strong contraction of the bladder wall.

It is very evident that the existence of active lower spinal segments has a profound influence on the behaviour of the detrusor muscle. In the presence of such active segments detrusor contractions similar to the normal continue to take place.

BEHAVIOUR OF THE SPHINCTER MECHANISM

1. Lesions of the Conus and Cauda Equina.—The sphincter mechanism consists of two essential parts, the one composed of smooth muscle and known as the internal sphincter, the other composed of striped muscle. The nature of the lesion leaves no room for doubt as to the behaviour of the latter. Since its innervation from the sacral segments of the spinal cord has been destroyed it must be entirely paralysed.

The evidence of cystography (*see Figs.* 513, 522, 526, 532) and of cystoscopy revealed that in every one of these cases there was wide relaxation of the vesical

orifice, and appeared to demonstrate complete incompetence of the internal sphincter.

The invariable presence of residual urine and the effort required to evacuate urine from the bladder showed that there existed in these cases a considerable resistance to emptying of the bladder. Since the external sphincter was paralysed and the internal sphincter incompetent it was necessary to find some explanation to account for this resistance. The only reasonable explanation seemed to be the existence of a mechanical factor—the pressure and elasticity of the tissues surrounding the urethra in the region of the triangular ligament. Dennig[2] came to a similar conclusion in the case of the dog after destruction of all three nerve pairs. Schwarz[10] also emphasized that the elastic action of the closing muscle is alone sufficient to maintain continence against a certain pressure.

When this work was first reported (Hunterian Lecture, Jan. 31, 1934) it was stated as a result of the above observations that the sphincter mechanism behaved entirely in a passive manner, and that there existed only this mechanical resistance to evacuation of the bladder. Denny-Brown and Graeme Robertson,[3] however, have placed on record observations which suggest that the internal sphincter plays an active part in bladder function in these cases. They state: "During spontaneous vesical activity the internal sphincter reacts by relaxations reciprocal with the intensity of contraction of the detrusor."

It appeared very difficult to reconcile the findings of Denny-Brown and Graeme Robertson with those above mentioned. But it seemed at least possible that the wide relaxation evident in the cystograms resulted from the technique employed, in which the skiagram was made immediately following the sudden injection of contrast medium. The problem was therefore further investigated. At the observations on March 9, 1935, in *Case* 1, a skiagram made at a time when no fluid was passing out of the bladder showed an open bladder neck and a widely dilated posterior urethra (*see Fig.* 516). In another film made three or four minutes after he had passed about 50 c.c. from the bladder no contrast medium can be seen in the posterior urethra (*see Fig.* 517). A second film made on the same occasion as *Fig.* 513 after the evacuation of a good deal of the fluid also showed a closed bladder neck (*see Fig.* 514).

Two important facts were demonstrated: (1) That even when the internal sphincter was widely dilated there was no escape of fluid; (2) That the internal sphincter does not always remain open.

A further series of observations was made (Sept. 18, 1935) in this case. After the introduction of fluid well-marked contractions of the bladder were evident, on one occasion the pressure increasing from 40 to 65 cm., on another from 38 to 65 cm. A film was made when the pressure had just fallen to 38 cm. following a spontaneous contraction. In this film there is wide relaxation of the vesical orifice and posterior urethra (*see Fig.* 518): 85 c.c. of fluid was then evacuated from the bladder and a film made with the pressure at 16 cm., and another when he attempted to pass urine (at a pressure of 38 cm.). In both these films (*see Fig.* 519) dilatation of the vesical orifice and posterior urethra is evident though very much less in degree than in the first film.

It is important to notice that the widest dilatation of the posterior urethra was present in the interval between the spontaneous vesical contractions. This does not confirm the interpretation given by Denny-Brown and Graeme Robertson[3] of

their observations on discharge into the urethra, according to which the internal sphincter should have been closed in the interval between the vesical contractions. On the other hand, the smaller degree of dilatation of the posterior urethra (seen after some of the fluid had been evacuated from the bladder—*Fig.* 519) was not made greater by the straining efforts to pass urine, although the film was made when the intravesical pressure was 38 cm.—the pressure at which a previous film had revealed the widest dilatation of the posterior urethra. This appears to be strong evidence in favour of the view that the condition of the smooth muscle sphincter is related to that of the detrusor, and that it is not simply a mechanical effect determined by the intravesical pressure.

In *Case* 3 the condition of the sphincter, as well as that of the detrusor, appears to have been different. In this case there was filling of the posterior urethra even when the greater quantity of the fluid had been evacuated (*see Figs.* 526–528). The degree of distension of the posterior urethra diminished as the volume of fluid in the bladder was reduced. The interpretation of this observation must be that the internal sphincter is incompetent and that with decrease of the intravesical pressure the size of the posterior urethra is diminished by the elastic action of the structures which surround it. Thus in this type of case the smooth muscle sphincter, just as the detrusor, becomes completely atonic.

In general it may be concluded from these observations that there is a considerable and purely mechanical resistance to the outflow of the urine from the bladder which must be attributed to the elasticity of the tissues in the region of the triangular ligament. There may be also closure at the vesical orifice itself, and the findings recorded here support the view of a reciprocal relationship between the detrusor muscle and the internal sphincter (Denny-Brown and Graeme Robertson).

2. Complete Transverse Cord Lesions.—The behaviour of the sphincter apparatus in such lesions has been carefully analysed by Denny-Brown and Graeme Robertson.[3] The only available evidence in this work is derived from *Case* 7. In this case the escape of fluid around the catheter during the pressure estimations, and the broad calibre of the urinary stream, testified to the fact that the urethral sphincters must have undergone a full relaxation in co-ordination with the detrusor contraction.

THE FUNCTION OF THE BLADDER AS A WHOLE

1. Lesions of Conus and Cauda Equina.—The remarkable degree of control which these patients were able to exercise clearly resulted from their ability, by straining efforts of the abdominal muscles, passively to force urine from the bladder. But in spite of the overwhelming importance of the abdominal straining as the expulsive force in urination it is certain that the urination is not entirely a passive procedure. It cannot be doubted that the stimulation to the detrusor muscle which results from the abdominal straining sets up some contraction of that muscle, which assists, if somewhat weakly, the expulsion of the urine. On one occasion in *Case* 1 abdominal straining had proved ineffective, but after pressure applied to the lower abdomen the desire to urinate occurred and further effort was successful. This shows that the activity of the bladder itself may be necessary in addition to the abdominal straining. In this instance it is to be presumed that the stimulation to the bladder wall set up detrusor contraction which in its turn

caused the internal sphincter to relax. Urination was thus facilitated when the patient made further efforts.

In spite of the degree of voluntary control which these patients can achieve, incontinence is a perpetual threat to their comfort. It is not probable that this is simply passive in character, from overflow. The sequence of events would appear to be that distension of the bladder to a certain point leads to sensation of bladder fullness, which is evidently transmitted by the hypogastric nerves. It is not unlikely that at this point a detrusor contraction is actually present, since Denny-Brown and Graeme Robertson's records show that the sensation of discomfort was related to contractions of the detrusor. At this point, too, the internal sphincter is no doubt widely relaxed, as was shown in a cystogram made in *Case* 1, where, although there was well-marked sensation, no fluid was escaping from the bladder (*see Fig.* 516). A little later, possibly as a result of a spontaneous contraction of the detrusor, or from some other cause such as a sudden movement or cough, some urine escapes from the bladder. It is important to notice that the amount of urine which escapes in this way at any one time is small, and in no way to be compared with the massive emptyings which result from deliberate urination. It is, indeed, only because the bladder is of itself so ineffective in expelling urine that the patient can control his urination to the degree described above.

2. Complete Transverse Lesions.—The behaviour of the bladder as a whole is well shown by an observation in *Case* 7 in which the urinary evacuations were watched for some time. Spontaneous evacuation occurred seven times as follows :—

1.	55	c.c.								
2.	95	c.c.	at	an	interval	of	35	minutes	after	1
3.	85	c.c.	,,	,,	,,	,,	16	,,	,,	2
4.	90	c.c.	,,	,,	,,	,,	10	,,	,,	3
5.	130	c.c.	,,	,,	,,	,,	$7\frac{1}{2}$	,,	,,	4
6.	130	c.c.	,,	,,	,,	,,	$8\frac{1}{2}$	,,	,,	5
7.	125	c.c.	,,	,,	,,	,,	8	,,	,,	the previous evacuation

Seven minutes after the last evacuation 100 c.c. of urine was found in the bladder, so that the amount of residual may be reckoned to be very small. Each evacuation seemed to be of normal force and calibre, and no urine escaped in the intervals. The features of this type of urination are, therefore, comparable to normal micturition—a powerful broad stream with almost complete emptying of the bladder. The fundamental difference from normal micturition is the entire absence of voluntary control. Incontinence is, therefore, absolute, and urinary evacuation takes place at intervals in a manner which may truly be described as " periodic reflex micturition " (Thomson-Walker[11]).

Case 7 always had a sensation from his bladder which preceded by a few seconds the actual evacuation and was evidently produced by the commencing activity of the bladder. This must have been transmitted by way of the hypogastric nerves. In spite, however, of the integrity of the latter he was not able to exercise any control over his bladder function.

The reflex facilitation of micturition from stimulations applied to areas below the level of the lesion have been studied by Head and Riddoch[4] and by Denny-Brown and Graeme Robertson.[3] Stimulation of the glans penis was shown to be effective in setting up reflex micturition in *Case* 7 and also in *Case* 8, the latter being, however, an incomplete lesion.

CONCLUSIONS

A study of the cases recorded in this work reveals quite clearly the fundamental importance to bladder function of active sacral spinal segments. The influence of these segments below a complete transverse lesion is such as to promote a perfect reflex micturition, which differs essentially from the normal in its entire independence of voluntary control.

The behaviour of the bladder in lesions of the conus and cauda equina is evidence of a limited degree of function mediated by the peripheral nerve ganglia. It has, however, been shown above that this function is not, of itself, efficient in evacuating urine from the bladder. For even when the internal sphincter is widely relaxed, and the detrusor in contraction, there may be no escape of fluid from the bladder. It can only be concluded, therefore, that its peripheral innervation alone determines a very ineffective bladder function.

It is without doubt the existence of a considerable mechanical resistance in the region of the triangular ligament which deprives this function of the bladder of most of its effect. But it is in virtue of this resistance combined with the patient's ability to expel urine by straining with his abdominal muscles which provides him with the means of living in relative comfort. This patient is, therefore, in much better circumstance than the patient with a transverse cord lesion who, though he has a very perfect reflex micturition, is not able to control it.

The author wishes to express his thanks to Professor Hugh H. Young, of Baltimore, and to Herr Professor E. Rehn, of Freiburg im Breisgau, for their courtesy in permitting him to study the cases under their care. And it is with especial pleasure that he records his great indebtedness to Mr. Geoffrey Jefferson for his continued interest and advice.

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A CASE OF SPINAL TUMOUR

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CHENCHIAH, a Hindu male aged 18 years, a ryot of Nellore district, was admitted to the General Hospital, Madras (under T. S.) on June 16, 1934, for inability to walk and pains in the back and sides of the chest of a year's duration.

HISTORY.—Nothing of clinical importance was noted. Prior to the present complaint, the patient was in perfect health, doing daily labour in the paddy fields. He is a teetotaller and never had any venereal infection.

About a year ago the patient noticed that he was progressively getting weaker in his legs. About two months later he could not stand without help and felt that the legs were giving way under him. At this time he suffered from sharp attacks of pain in the middle of his back and radiating to both sides of his chest. The weakness in the lower limbs steadily progressed, and in the last three months prior to admission he was unable to stand up even with assistance. Later any attempt to move the legs resulted in involuntary contractions. Sometimes this occurred spontaneously. A month ago he had retention of urine and fæces. He was catheterized in a hospital and later sent to the General Hospital.

ON ADMISSION.—The patient was rather poorly nourished, slightly anæmic, and had moderate pyorrhœa. Cardiovascular, respiratory, and alimentary systems were normal. No abnormal changes were seen in blood, urine, or motion.

Neurological Examination (T. S.).—Mental powers were normal and unimpaired. Both ocular fundi were normal. No abnormal changes were seen above the chest.

Musculature.—Lower limbs: Some wasting of muscles with occasional flexor spasms was seen. Motor power was considerably diminished in both limbs, the weakness being more marked in the left. Both legs were very spastic. The muscles of the face, neck, upper limbs, and trunk were normal.

Sensory.—Sensations in the lower limbs were moderately diminished. The loss of proprioceptive sensations was more marked than the others in the left leg, but thermal and pain sensations were diminished more in the right leg. Appreciation of light touch was impaired on the trunk up to the 8th dorsal segment on the right and 10th dorsal segment on the left. Girdle pains were present corresponding to the 10th dorsal segment, i.e., on a level with the 7th–8th dorsal vertebræ.

Reflexes.—These were as follows:—

	Right Lower Limb	Left Lower Limb
Babinski	+	+
Cremasteric	—	—
Abdominal, lower	—	—
Abdominal, upper	±	±
Knee-jerks	+ +	+ + +
Ankle	+ +	+ + +
Patellar and ankle clonus	+	+ +

Organic reflexes: Bouts of obstinate constipation and retention of urine occurred.

Electrical Reactions.—No reaction of degeneration was noted in the muscles of lower limbs.

Other Investigations.—Roentgenogram of vertebral column was negative. Wassermann and Kahn reactions were negative.

Lumbar Puncture.—Fluid was under pressure, pale yellow but clear. Delayed spontaneous coagulation occurred. Total proteins, 890 mgrm. per cent; albumin, 554·6 mgrm. per cent; globulin, 325·4 mgrm. per cent; lymphocytes, 3 per c.c. Gold curve and Wassermann reaction of fluid were negative. Clearly it was a case of Froin's syndrome.

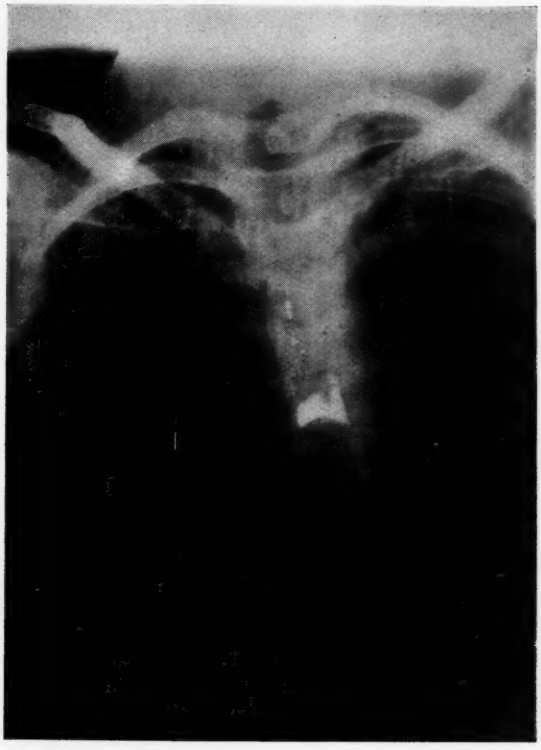

FIG. 541.—Antero-posterior view of spinal column after injection of lipiodol.

On July 24 a cisterna puncture and injection of lipiodol was done by N. M. R. and on July 27 a lumbar puncture and injection of 1 c.c. of lipiodol (heavy) in high Trendelenburg position was done and roentgenograms were taken (*Fig.* 541). They showed a definite block opposite the 7th dorsal vertebra. The case was transferred to the surgical side under N. M. R.

OPERATION (Oct. 16, by N. M. R.).—Incision from the 3rd to 10th dorsal spines was made, the spines were exposed and removed from the 5th to 9th inclusive. The laminæ of the 7th and 8th dorsal vertebræ were trephined and the edges were nibbled away. The dura was exposed and lifted by sharp hooks followed by catgut sutures. The dura was incised for about 2½ in. in the middle line, exposing an œdematous arachnoid with a small yellow calcareous-like nodule at the lower part. There was no visible pulsation. In the lower part of the wound

the pale yellow-coloured cord could be seen. The arachnoid was incised carefully in the upper part, exposing a vascular tumour. This was slowly shelled out. It occupied the left dorso-lateral position in the theca and extended slightly to the right across the mid-line. The tumour was not attached to the dura but was entirely in the arachnoid tissue. After this the cerebrospinal fluid with some lipiodol escaped from above and visible pulsation became evident.

After the usual toilet the theca was closed with fine silk and the wound closed in layers. The patient was nursed in the prone position and put on hexamine and injections of cylotropine. Except for a small irregular rise in temperature his

FIG. 542.—Roentgenogram after the operation showing most of the lipiodol at the bottom of the theca.

condition was good and the wound healed by first intention. He began to improve, and by Nov. 28 he was able to move about without aid. On Nov. 12 a lumbar puncture showed 300 mm. pressure, and 20 c.c. of fluid were withdrawn. The cerebrospinal fluid showed 32 mgrm. per cent of albumin and a trace of globulin; the cell count was about normal. On Nov. 19 another lumbar puncture showed 240 mm. pressure, and 20 c.c. were withdrawn. After this his progress was steady. *Fig.* 542 shows the spinal condition after operation.

REPORTS OF EXAMINATION (T. S.).—

Nov. 10, 1934.—Increased strength in legs; flexor spasms and root pains absent.

Nov. 28, 1934.—Patient is able to walk about without aid; gait still slightly spastic; Rombergism is greatly diminished. Sensory changes and signs of damage to pyramidal tract persist.

Feb. 1, 1935.—General nutrition is very good. The patient is able to walk about very freely with just a trace of stiffness. Light touch and kinæsthetic sensibility are not completely recovered. Rombergism and root pains are absent. In so far as the damage to the pyramidal tract is concerned the deep jerks are still a little 'gay' on both sides and the superficial skin reflexes are still absent. This is probably due to some permanent damage caused by the tumour.

FIG. 543.—Painting of a vascular fibroma weighing 45 gr. Site: dorso-left-lateral position over the 8–9th thoracic segments, in the arachnoid, but not attached to the dura. (×2.)

FIG. 544.—High-power photomicrograph of section of tumour.

The patient is able to run and turn sharply round without stumbling and appears normal except for the slightly exaggerated tendon reflexes in the legs and absence of skin reflexes.

He was discharged quite happy and pleased on March 17, 1935.

The tumour was fairly vascular and weighed 45 gr.

PATHOLOGICAL REPORT.—Cellular fibroma which is very vascular; areas of hæmorrhage are also seen. (*Figs.* 543, 544.)

We are obliged to the Barnard Institute of Radiology for the roentgenograms, to the Professor of Pathology for the painting and photomicrographs, and the Professor of Biochemistry for reports on the cerebrospinal fluid.

A CARCINOID TUMOUR OF THE LOWER ILEUM

By W. QUARRY WOOD, Edinburgh

Carcinoid tumours of the gastro-intestinal tract have excited a great deal of interest among pathologists during the past twenty years, an interest which is perhaps out of proportion to their clinical importance. They are rare tumours, but in their origin, structure, and method of spread they present peculiarities which are responsible for the amount of attention they have attracted. They are most common in the appendix, but have been recorded as occurring in all parts of the abdominal alimentary canal. The cases recorded in the small intestine up to the latter part of 1934 numbered 152; since then a few additional cases have appeared in the literature. The comparative rarity of the tumour and the fact that it is now being recognized that a rather characteristic group of symptoms are frequently associated with the condition may justify the publication of another case.

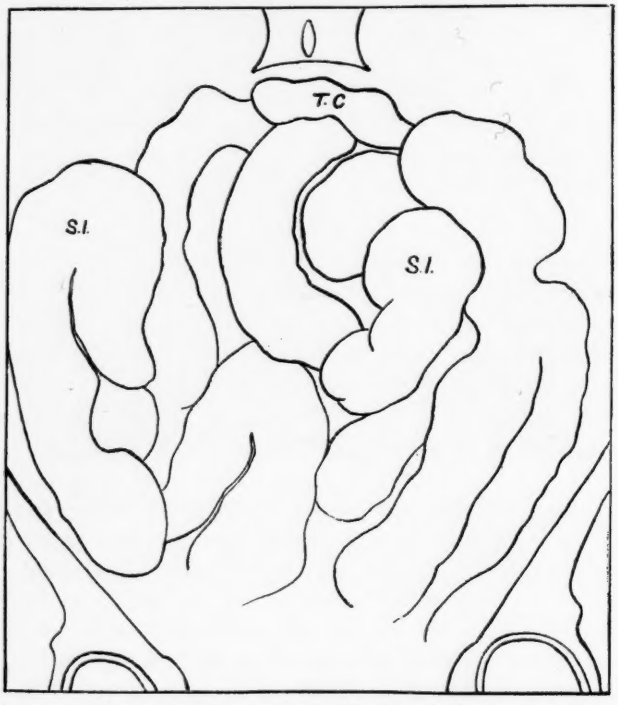

Fig. 545.—A tracing of the X-ray photograph showing distended coils of small intestine (S.I.). A small amount of barium has entered the transverse colon (T.C.).

CASE REPORT

A woman, aged 62, had an attack of abdominal pain and flatulent distension in the spring of 1933, a year before operation. She was free from symptoms during the summer, but in the late autumn attacks of pain and flatulence recurred. In the beginning of January, 1934, the attacks became more severe and she was much troubled with constipation. She lost a stone in weight during the last few months before operation. Her symptoms suggested

chronic intestinal obstruction probably due to a carcinoma of the colon. X-ray examination, however, showed that the site of the obstruction was at the lower end of the small intestine (*Fig.* 545). On March 18 the abdomen was opened through a right paramedian incision and a tumour was found at the ileocæcal junction. This was regarded as a carcinoma. The small intestine was much distended and an immediate resection was considered to be unsafe. The ileum was accordingly divided about 6 in. above the ileocæcal junction, and the proximal

FIG. 546.—The appearance of the tumour on section.

end united to the transverse colon by a lateral anastomosis. On April 15 resection of the terminal ileum, cæcum, and ascending colon was carried out. The patient made a good recovery from the operation and when seen on Nov. 24, 1935, was found to be in excellent health.

The appearance of the specimen is well shown in *Fig.* 546. The cut surface of the tumour presents a yellowish colour which was very striking in the fresh condition. It is seen to be growing from the upper border of the terminal part of the ileum about 2 cm. proximal to the ileocæcal valve. It forms a flattened cake in the position of the mucous,

submucous, and muscular coats, and has become buckled on itself owing to contraction of its basal part. This has caused it to form a rounded projection which is obstructing the lumen of the bowel. The tumour tissue can be seen to extend through the muscularis. The ileum proximal to the growth shows a well-marked degree of hypertrophy and dilatation. The bowel distal to the tumour is normal in appearance except that the ileocæcal valve is unduly prominent. The orifice of the appendix is occupied by a probe. The appendix is normal in appearance, showing no evidence of either inflammation or tumour.

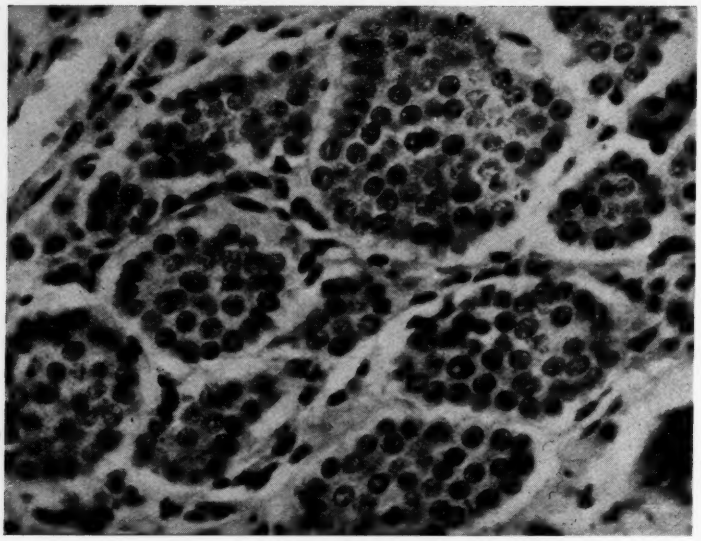

FIG. 547.—The microscopical appearance of the tumour.

The microscopical appearance of the tumour is seen in *Fig.* 547. A report by Colonel W. F. Harvey states: "The condition is a typical carcinoid tumour of spheroidal-cell type. It presents solid alveolar masses of a uniform-cell type, situated in the submucosa, the muscularis, and in the subserosa. The tumour growth in this case is much more extensive than those one has seen." One lymphatic gland was found about 2·5 cm. above the growth; it was the seat of a metastatic deposit. A small nodule of doubtful nature was found in the mesentery adjacent to the tumour; this consisted of tumour tissue, but it was uncertain whether it represented an infected lymphatic gland or not.

DISCUSSION

The most striking features of the carcinoid tumour are the yellowish colour of the cut surface on naked-eye examination and the presence on microscopical examination of silver-reducing granules in the cytoplasm. The cells of the tumour are frequently arranged in solid columns, resembling certain types of basal-cell carcinoma. The histogenesis of the tumour has aroused much speculation. Argentaffine cells, the so-called Kultschitsky cells, are normally present in the crypts of Lieberkühn; they are concentrated at the bases of the crypts, being pear-shaped with broad bases lying on the basement membrane and with narrow bottle-shaped necks leading up to the lumen of the crypt. The cytoplasm appears lighter after staining than that of the neighbouring cells. They are found in all parts of the intestinal tract but are most numerous in the appendix and terminal ileum.

Their origin is uncertain. Raiford[1] suggests that they are probably of ectodermal origin and have migrated early from the neural crest to form a specialized part of the chromaffin system. The affinity for silver and the similarity to the cells of the adrenal which they present are in favour of this view. Most pathologists have followed Masson[2] in attributing the origin of carcinoid tumours to these cells. Raiford makes the following statement: "It is ascertained, beyond reasonable doubt, that the carcinoid tumours arise from the cells of Kultschitsky or the chromo-argentaffine cells of Masson and that these cells are normally present in the intestinal tract of man. They are thought to be chromaffin in nature and the tumours are consequently chromaffin tumours. Their significance, or the stimulus resulting in tumour formation, can at best remain a matter of speculation." Masson also demonstrated a peculiar relationship between carcinoid cells and nerve-fibres, said to resemble the relationship between nerve-fibres and nævus cells. The carcinoids were first described and separated from the carcinomas by Obendorfer[3] in 1907.

From the clinical point of view, most interest lies in the nature of the symptoms which are associated with the tumour and in its degree of malignancy. In carcinoid tumours of the appendix metastasis to the regional lymphatic glands is extremely rare and distant metastasis is practically unknown. In the small intestine the tumours are much more malignant, secondary growths having been frequently met with in the regional lymphatic glands and occasionally in the liver, the lungs, and other organs. A survey of several of the recently recorded cases of carcinoid tumour of the small intestine shows that a characteristic group of symptoms may sometimes be present and that a pre-operative diagnosis of the probability of the lesion being a carcinoid might be possible. A brief reference to a few of the recent cases is of interest. The case reported by Christopher[4] occurred in a woman of 55. The tumour was situated 8 in. proximal to the ileocæcal junction. It had caused recurrent attacks of abdominal pain over a period of three years with a mild degree of intestinal obstruction. A local resection of the affected piece of bowel was followed by recovery. No metastases were found in the regional lymphatic glands. Two cases reported by Lee and Taylor[5] resemble my own case very closely. In one, a woman aged 51, attacks of abdominal pain associated with loss of weight and constipation had occurred over a period of thirteen months. A tumour was found at the ileocæcal junction, and resection of the terminal part of the ileum, the cæcum, and the ascending colon was performed. The tumour had extended through the muscular coat of the bowel and one lymph gland was almost entirely replaced by a metastatic growth. This patient was free from abdominal symptoms twenty-three months later. The second case was also in a woman, who was 53 years of age. Similar symptoms had been present, but only for a period of one month. The tumour in this case was 10 cm. proximal to the ileocæcal valve and a similar operation was carried out. This tumour had also spread through the muscularis and had infected one of the neighbouring lymphatic glands. The patient was well twenty-two months later. It is of interest that these three patients and my own were females of about the same age.

The clinical result of a carcinoid tumour of the small intestine is likely to be a slowly progressing stenosis of the bowel. This is borne out by a consideration of the above cases and by a survey of the literature. In a patient after middle age presenting symptoms of chronic intestinal obstruction, where the radiological

examination shows the site of the lesion to be the lower part of the small intestine, the possibility of its being a carcinoid tumour should be considered. An adenocarcinoma will present similar symptoms and it will generally be impossible to differentiate between the two until the tumour has been sectioned. The distinction is of some importance in view of the lesser degree of malignancy of the carcinoid. In the past, however, an unjustifiably optimistic view seems to have been held in regard to the essential nature of these tumours, probably owing to the fact that in the appendix the carcinoid is almost invariably benign. The probability is that all carcinoids are potentially malignant. In the appendix the lesion produces symptoms at an early stage before infiltration has progressed very far. It is to be noted in this connection that the age incidence in carcinoid of the appendix is much lower than in carcinoid of the intestine; in the appendix the average age has been under 30 in several series of cases, while in the small intestine it is between 50 and 60. In the small intestine the tumour reaches a larger size before it gives rise to symptoms, and in these larger tumours infiltration of the muscularis and the serosa are not infrequently present; metastases in the regional lymphatic glands and occasionally in the liver, the lungs, and other organs are sometimes met with. Malignancy seems to be largely a question of the age of the tumour. In the 152 cases collected by Humphreys[6] at the end of 1934, metastases were present in 24·4 per cent. With the addition of 3 out of the 4 cases mentioned in this article the frequency of metastases becomes 25·9 per cent. It is significant that metastases have been found more frequently in the recent cases than in those reported earlier. As compared with adenocarcinoma, the carcinoid tumour is less malignant, but the difference is not so great as has been commonly supposed. The frequency of metastasis in adenocarcinoma of the small intestine is given as 38 per cent by Schleips[7] and as 36 per cent by Craig.[8] It is obvious that the treatment of a carcinoid tumour of the intestine must be a radical operation on the same lines as in adenocarcinoma.

Reference must be made to a further feature of carcinoid tumours. In cases discovered at post-mortem examination the tumours have been found to be multiple in a considerable proportion of cases—probably about a third. In these multiple cases many of the nodules have been of small size and at the 'benign' stage. It will very seldom be possible to recognize or to deal with such multiple growths in the living subject.

I wish to express my indebtedness to my wife for the coloured illustration of the tumour, to Colonel W. F. Harvey for his help in the preparation of this paper, and to Mr. Hamilton for the photomicrograph of the growth.

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COLLOID TUMOUR OF THE URACHUS INVADING THE BLADDER

By R. CAMPBELL BEGG, Wellington, New Zealand

In 1931 I published in this Journal a case of colloid adenocarcinoma of the urachus invading the bladder vault, and expressed the opinion that the seventeen cases reported in the literature up to that time in no way represented the frequency of the disease. I am now able to report a second case of a similar nature.

W. J., male, aged 54 (Wellington Hospital Case No. 34/5985) was admitted on March 19, 1934, complaining of passing blood in the urine.

History.—The patient said that two years previously he had received a violent blow in the back and two months thereafter passed a large quantity of blood in his water. The bleeding was apparently profuse and accompanied by clots. Ever since then at intervals of about seven weeks he had had recurrent attacks of a similar nature. These usually lasted about two weeks, but in the intervals, and especially in the few months before admission, he had passed globular masses of a material resembling apple jelly. While during the attacks he was annoyed with frequency, especially at night, and occasional pain on passing clots, he had no symptoms whatever in the intervals. There was nothing of importance in his previous history.

Fig. 543.—Cystoscopic appearance of bladder invasion by colloid adenocarcinoma of the urachus. The reddish neoplasm is seen shining through a mass of translucent jelly at the bladder apex. The surrounding mucosa is normal.

On Examination.—The patient appeared to be a trifle anæmic, but the general examination disclosed no physical anomaly. Neither kidney was enlarged, palpable, or tender. There was slight tenderness on deep pressure in the suprapubic region. The prostate was normal to rectal touch and no induration was felt at the base of the bladder. The Wassermann reaction was negative. The urine contained much mucin, many blood and large epithelial cells, and a few hyaline casts.

Cystoscopy.—The bladder and ureteric orifices were normal with the exception that adherent jelly-like masses were present in the bladder vault somewhat obscuring the view. It was seen, however, that these masses of jelly adhered to a projecting and nodular tumour, the circumference of the base of the growth being about equal to that of a penny (*Fig.* 548).

A diagnosis was made of colloid adenocarcinoma of the urachus invading the bladder apex; operation was advised but declined, the patient being discharged. He was readmitted for operation on Jan. 20, 1935; this was performed on Jan. 22.

OPERATION.—The radical operation advocated as the result of recurrence in the transversalis-peritoneal space in my previous case was carried out—namely, the removal of the umbilicus and the surrounding skin, a wedge-shaped area of peritoneum and transversalis fascia, and the upper half of the bladder.

An incision was made for ¾ in. around the umbilicus and deepened above and at the sides until the peritoneal cavity was opened. The incision was continued in the middle line as far as the symphysis pubis, and the aponeurosis of the recti muscles incised and the muscles retracted. An incision was then made through the peritoneum and transversalis fascia on each side as far out as the course of the obliterated umbilical arteries. The umbilicus was held upwards and forwards and the peritoneo-transversalis flap turned forwards until the posterior surface of the bladder came into view. Without making any inspection of the growth the posterior wall of the bladder was cut transversely with the endothermy knife and with some separation of the tissue laterally wide of the bladder, the incision was carried round the organ, removing the upper half of it together with the fat and fasciæ on its superior surface. Some masses of colloid material lying loose in the remaining part of the bladder were removed and the walls brought together leaving a small suprapubic Malecot catheter for drainage. The peritoneum was mobilized, the edges brought together, and the aponeurosis and skin sewn. Following this somewhat extensive operation three was a considerable amount of post-operative distension which was overcome with the use of acetylcholine.

FIG. 549.—Sagittal section of the urachus and the bladder apex. The whitish tumour is seen to invade the urachus uniformly without infiltrating the surrounding tissues. It passes cleanly through the bladder musculature without invading it. The recurved striated appearance of the lower end is apparent.

SUBSEQUENT PROGRESS.—The patient made a good recovery and was discharged on Feb. 2. At that time, with the exception of slight frequency due to the diminished size of the bladder, he had no trouble in micturition and the urine was clear. He reported again in June, five months later. Micturition had become normal and there was no evidence of recurrence in the bladder or elsewhere.

PATHOLOGY.—The specimen consisted of the umbilicus and surrounding skin, a wedge of the peritoneum and transversalis fascia, and the upper half of the bladder with the surrounding fat and fascia. Viewed from below the projecting nodular tumour with a mass of colloid the size of a golf ball was seen. The mass was cut sagittally and it was found that the growth occupied the urachus and extended as a cord, following this organ down in its intramural course through the bladder, spreading out in a fan-shaped manner on the vesical mucosa (*Fig.* 549).

Sections of the urachus above the bladder level showed that the whole of the urachal epithelium had been converted into tumour but without invasion of the fascio-muscular wall (*Figs.* 550, 551).

The growth itself at no point showed a tendency to invasion and was of a papillomatous nature rather than acinar. Papillæ with definite cores were seen, but instead of being covered with transitional many-layered epithelium the cells were tall columnar in type with basal nuclei and many goblet cells with a copious secretion of mucin between the papillæ. They were close set and resembled intestinal villi. In spite of the absence of invasion of the cells this growth must be classed as colloid carcinoma, though the term 'adenocarcinoma' would not apply on account of the absence of acini, and the papilliform nature of it was most unusual for a colloid growth. Abnormal types of growth which can indeed pass from one type into the other are, however, to be expected in this neighbourhood,

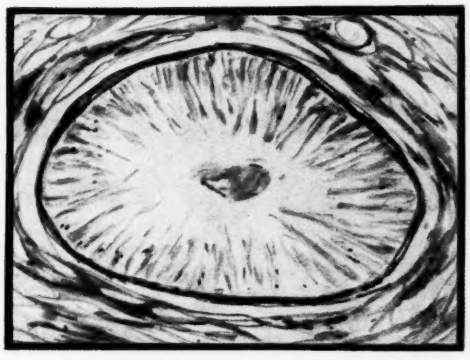

FIG. 550.—Lens view of section of the urachus 1 cm. above the bladder. The delicate papillomatous-like fronds have replaced the normal urachal epithelium, but the urachus has retained its normal oval appearance. A small core of colloid material lies in the centre of the lumen. (× 5.)

FIG. 551.—Low-power microscopic view of a small portion of the wall of the urachus. It can be seen that each papilliform structure consists of a fibrous-tissue core bearing a blood-vessel. The epithelium, however, is of the long columnar type with basal nuclei. Many of them are goblet cells full of mucin. The strong resemblance to a section of intestinal mucosa is striking.

the epithelial debris and its cells being more or less totipotent in character—the general tendency being towards intestinal forms.

The question of the origin of these colloid tumours and the necessity for being aware of their occurrence and appearance as apical bladder growths was so fully discussed in the previous article that there is little to add. Their rarity, it is true, makes their study of somewhat academic interest, and yet two have occurred in my own practice in the course of ten years, both from the comparatively small city of Wellington. To attain even a probability of cure the treatment must differ from the usual one of resection of the bladder. The symptom of hæmaturia combined with the passing of much jelly-like mucus in the urine is almost pathognomonic, though I have encountered one case of colloid carcinoma of the base of the bladder, the more interesting because it was accompanied by pseudomyxoma peritonei. Faced with such symptoms a primary growth of the urachus may be postulated, and the only appropriate procedure is the radical operation

recommended in the previous paper and carried out in the case now described. The danger of recurrence is not in the bladder but in the space between the transversalis fascia and the peritoneum between the apex of the bladder and the umbilicus.

For the coloured illustrations I am indebted to Dr. P. A. Treahy, Assistant Urologist in the Wellington Hospital Service.

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TWO CASES ILLUSTRATING THE LATENCY OF LARGE RENAL CALCULI AND THEIR RELATION TO PREGNANCY

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THE extreme latency of some urinary calculi is illustrated by the two following cases. In one a perinephric abscess occurring during the puerperium of an eighth pregnancy gave the first hint of serious renal dysfunction; in the other impaction of a small stone in the pelvic outlet was associated with threatened anuria and brought to notice a most interesting collection of stones in the opposite kidney and ureter, the existence of which had not been previously suspected.

Case 1.—Mrs. K. S., aged 36 years, was admitted to Birkenhead Municipal Hospital on July 1, 1935, with a diagnosis of renal neoplasm. Six weeks prior to

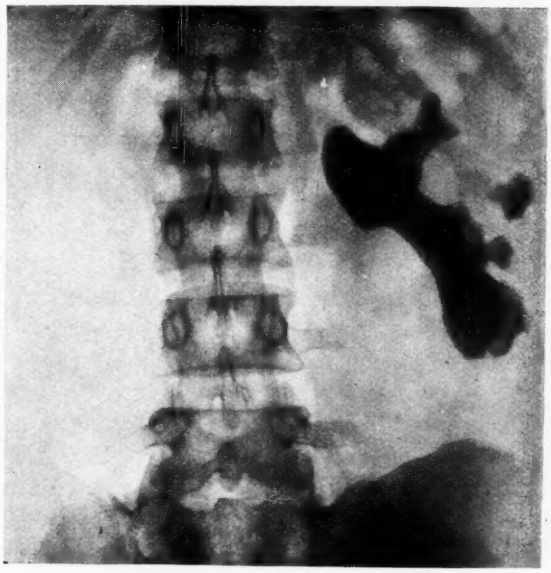

FIG. 552.—*Case* 1. X-ray appearances.

admission she had been delivered of her eighth child. Apart from loss of weight and anorexia dating back some ten months, the patient had been entirely symptomless. A slight albuminuria had complicated the last pregnancy. A stone had been removed from the left kidney in 1915. On June 28 a dragging pain was experienced radiating from the left loin to the pubis. There was no frequency or dysuria.

ON EXAMINATION.—A large slightly mobile mass was found occupying the left kidney region. This mass was irregularly nodular and considerable pain was experienced on bimanual examination. The temperature was 100·8° and the pulse 130. The urine was acid and contained albumin, blood, and pus. The specific

gravity was 1020. The blood-urea was 43 mgrm. per 100 c.c. and the blood N.P.N. 40 mgrm. per 100 per c.c.

The diagnosis of a large branched phosphatic stone was made, and the presence of this stone was confirmed by X-ray examination (*Fig.* 552). In view of the clinical findings, a left-sided pyonephrosis was considered a probable complication of the stone.

OPERATION (July 8).—The left kidney was exposed through the usual lumbar incision. It was adherent, especially posteriorly along the line of the previous incision, and there were marked fibro-fatty changes in the perinephric fat. A small perinephric abscess was found in relation to the lower pole of the kidney, but there was no obvious communication with the kidney, the spread of infection being presumably a lymphatic one. There was a marked pyonephrosis. Nephrectomy was performed and on completion of the operation a pint of compatible blood was transfused. A rubber tissue drain was inserted.

Recovery was uninterrupted, blood and pus disappearing from the urine after two days and albumin five days later.

The chief interest in this case is the post-partum flare-up of the renal infection and the fact that such infection did not occur until the eighth pregnancy, or rather that not until a perinephric abscess (one of the rarer complications of renal stone) formed, did the patient's symptoms call for serious investigation.

Case 2.—Mrs. L. D. aged 42 years, was admitted to Birkenhead Municipal Hospital on Aug. 19, 1935, in a state of threatened anuria. The history given was that since August, 1934, a severe right-sided renal colic had recurred at monthly intervals. On Aug. 15, 1935, urinary frequency preceded an attack of right-sided colic, and since then only a few ounces of urine had been passed daily. The patient would admit to only one attack of left-sided renal pain, which was experienced on the day prior to admission to hospital. In June, 1931, while recovering from a left-sided hemiplegia she was delivered of her sixth child. Apart from these six full-term confinements she had had two miscarriages. Her last menstrual period occurred on July 20, 1935.

ON EXAMINATION.—There was marked tenderness on bimanual palpation of the right kidney with cutaneous hyperæsthesia over the line of the right ureter. An indefinite mass was palpable in the left iliac fossa. The urine, which was very scanty, contained albumin, blood, and pus, and gave a culture of *B. coli*. The blood-urea was returned at 117 mgrm. per 100 c.c. and the blood N.P.N. at 85 mgrm. per 100 c.c.

X-ray examination after the administration of uroselectan B showed stones in the left kidney and a large stone in the left ureter (*Fig.* 553). The left kidney appeared very large and hydronephrotic. The right kidney excreted a little uroselectan, but none of it appeared in the ureter, which was jammed by a single stone. The right kidney function appeared to be poor.

FIRST OPERATION (Aug. 22).—A right-sided pyelotomy was performed and the stone removed. The kidney was hydronephrotic. The incision in the pelvis was not sutured, but a rubber drainage tube was passed down to the opening.

Thereafter the patient's general condition rapidly improved and there was a free excretion of urine. On Aug. 23 the blood-urea was 61 mgrm. per 100 c.c. and the N.P.N. 58 mgrm. per 100 c.c. On Sept. 2 the blood-urea was 44 mgrm.

per 100 c.c. and the N.P.N. 42 mgrm. per 100 c.c. By Sept. 6 the renal function had ceased to cause anxiety, but on the evening of that day the patient had an incomplete abortion which was completed at 7.30 a.m. on Sept. 7. Her condition was once more critical, but she responded well to a transfusion of compatible blood, and was discharged from hospital on Sept. 23 in good health.

She returned to hospital on Oct. 23. The urine contained some albumin and many leucocytes and gave a culture of coliform bacilli. The blood-urea was 35 mgrm. per 100 c.c. and N.P.N. 45 mgrm. per 100 c.c.

SECOND OPERATION (Oct. 28).—Under percain anæsthesia the left kidney and greater part of the left ureter were removed through a lumbar incision carried

FIG. 553.—*Case* 2. X-ray appearances.

forward parallel to the crest of the ilium. The ureter was mobilized with comparative ease and the only real difficulty encountered during the operation was due to a somewhat thickened renal pedicle. Convalescence was uneventful.

The ureteric stone weighed approximately 50 grm. The length along the curved axis was 5 in., net length $4\frac{1}{8}$ in., and the maximum circumference, near the lower end, was 4 in. The circumference of the stone in the left kidney was approximately $\frac{3}{4}$ in.

Section of the kidney showed some cloudy swelling and loss of cell outline, with a few patches of round-celled infiltration.

I wish to thank Mr. R. A. Grant for operating facilities, and the staff of Birkenhead Municipal Hospital for their help in investigating these cases.

THREE CASES OF DUODENAL DIVERTICULUM REMOVED BY OPERATION

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Two kinds of diverticula are found in the duodenum: (1) Primary; (2) Secondary. The latter are due to traction of some neighbouring inflammatory process such as an ulcer, and invariably occur in the first part of the duodenum. They are somewhat rare. The primary diverticula, with which we are now concerned, are much more common. They never arise in the first portion, are seen occasionally in the third and fourth portions, but in the large majority of instances (75 per cent) are found springing from the concave inner aspect of the descending limb, never far from the ampulla of Vater, and lying in close relationship to the head of the pancreas.

Their structure is frail: when dissected out from the areolar tissue which surrounds them they are collapsed, thin-walled, and consist of the mucous and submucous coats of the intestine with perhaps a few muscle fibres gradually fading away as the flask-shaped fundus is reached. Their opening into the bowel varies in size and may be large enough to admit the tip of a finger. There would seem, therefore, to be little hindrance to the entrance and exit of chyme, though the deficiency of muscle fibre in the wall would favour stasis within the sac.

These diverticula are herniæ of the duodenal wall, and the close relationship of their necks to blood-vessels which pierce the concave aspect of the bowel, or to the entry of the common bile-duct, suggests a gradually developing protrusion through a weak spot thus formed. Thus, although occasionally discovered in the young, they are more and more frequently found as age advances. A very full consideration of developmental theories, as indeed of all aspects of the subject, is to be found in Odgers's[1] classical paper and more recently in one by Edwards.[2]

Duodenal diverticula may be solitary or multiple—the former in large preponderance. Their incidence is variously computed by different investigators. Odgers quotes statistics obtained from the post-mortem room, which vary from 3·3 per cent (Linsmayer) to as high as 14·2 and 16 per cent (Baldwin and Grant), and others from X-ray investigations which range from 1·18 per cent (Andrews), 1·2 per cent (Case), 1·5 per cent (McMullan), 3·8 per cent (Spriggs and Marxer), to 5·19 per cent (Cryderman), the five latter giving an average of 2·5 per cent over 11,470 examinations. It is evident, therefore, that as an anatomical entity, duodenal diverticulosis is no rarity. But when we consider its possible clinical importance, we find great differences of opinion.

Rare instances of inflammation or even of perforation of the sac have been recorded, and others of cholangitis or pancreatitis in which the close association of a diverticulum would appear to have been a factor in provoking stasis and inflammation in the respective ducts. Apart, however, from these rarities, the vast

majority of pouches removed by operation or post-mortem, when examined microscopically, show no trace of inflammation past or present, and it is hardly surprising to find scepticism as to whether an uninflamed diverticulum *can* give rise to clinical symptoms. Probably it seldom does. Nevertheless, instances occasionally occur in which it would appear to do so.

Let us take the case of a patient with prolonged and troublesome digestive symptoms conforming to no special type, in whom a duodenal pouch has been found in the course of a bismuth meal and X-ray examination. If other possible causes have been carefully examined, and as far as possible excluded, if medical and dietetic treatment have been tried for a reasonable period without permanent benefit, and particularly if stasis within the pouch has been shown to be present, are we not, under these conditions, justified in regarding it as a causal feature and in recommending an operation? The three cases which we present support such a view. Happily in two, both pure and uncomplicated by other surgical procedure, the relief from symptoms was absolute. One of these has remained well for a year and nine months and the other for eight years. In the third case amelioration was so great as to give the patient the utmost satisfaction, but, as will be seen from the notes given below, it cannot be counted as a pure case, in that a pyloric adhesion was a possible alternative or additional factor in the causation of symptoms.

In no case did the operation itself present any special difficulty, but in this perhaps we were fortunate, for in each instance the neck of the sac was sufficiently distant from the common bile- and pancreatic-duct entrances to give a clear field for removal and suturing, nor were there any adhesions. But it is evident from the descriptions of Odgers[1] and Edwards[2] that in some cases operation may be difficult—e.g., where the pouches are multiple, or adherent—or indeed impossible, as when one or more have developed in direct relationship to the ampulla of Vater.

CASE REPORTS

Case 1.—Mrs. L., aged 64. Admitted to the Royal Sussex County Hospital under H. N. F., on Feb. 8, 1927, complaining of pain in the upper abdomen to the right of the mid-line—five months since onset.

HISTORY.—The pain occurred frequently, in attacks which lasted an hour or more, and which were accompanied by much eructation of wind. She described the pain as a constant, deep ache, severe enough to make her feel faint. It might come on any time and bore no special relation to food. Her appetite was poor and there was always a good deal of gastric flatulence after meals. The bowels were constipated and an action sometimes brought on the pain or made it worse. Micturition was normal. She had lost weight lately.

ON EXAMINATION.—There was nothing special to note in the abdomen except the scar (right paramesial) of an operation for a gangrenous appendix five years previously, and a point tender on pressure one inch to the right of the mid-line, midway between umbilicus and costal margin.

X-ray Examination..—A barium enema revealed no filling defect but caused pain during entry, and there was marked spasm of the transverse colon. A barium meal showed entirely normal behaviour and appearances of the stomach and duodenum, and there was nothing to note in the passage of the meal through the intestine. Barium, however, lodged in a diverticulum, the shadow of which was bounded by the pylorus and the three parts of the duodenum. There was marked tenderness over this pouch on palpation. The position of the stoma could not be identified. Barium remained in it after the rest of the meal had passed through the duodenum.

In view of the protracted pain, discomfort, and loss of weight, it was decided to explore the abdomen.

OPERATION.—Right paramedial incision. Biliary system, pancreas, stomach and duodenum, right kidney, and colon, all appeared normal. A few minor adhesions in the cæcal region were left undisturbed. There was no sign of a diverticulum on the anterior surface of the duodenum. The latter, after incision of the peritoneum to its right, was mobilized and turned over to the left. The pouch was found bulging through the small arcade of vessels supplying the duodenum on its concave surface and was carefully teased out through a small gap in them, till, with gentle traction on the fundus, a purse-string suture could be placed round the rather wide stoma. The neck was clamped, divided, and ligatured and the circular suture tied. The lateral peritoneum was closed and the abdominal wound sutured.

The position of the stoma was on the postero-inner aspect of the duodenum and apparently above the entry of the common bile-duct. The latter was not seen. The pouch was 4 cm. in length, delicate, and thin-walled. Microscopically it showed a mucosal lining with a few muscular fibres thinned out to practically nothing at the fundus. There was no evidence of inflammation, old or recent, in the sac.

SUBSEQUENT PROGRESS.—The patient made a good recovery and was entirely relieved of her pain and flatulence. She has been seen from time to time during the last eight years—on the last occasion, one month ago—and she has kept in excellent health.

Case 2.—Miss M. H., aged 52. Intractable recurrent vomiting for twenty years. Single diverticulum demonstrated by X-rays. Laparotomy. Removal of diverticulum. Complete cure up to one year and nine months after operation.

HISTORY.—Miss M. H. (under care of L. I. M. C.) complained of attacks of intractable vomiting during which everything taken into the stomach was rejected. The sequence of events was that after a period of complete comfort, the patient would experience a feeling of oppression high in the epigastrium and below the right costal margin, acid eructations, bringing up of wind, and slight nausea. Vomiting commenced soon after, and persisted day and night for three or four days in spite of treatment, with special tendency to occur about half an hour after food. In the last three years the syndrome was less related to the taking of food and in addition a favourite time of onset was between 2 and 3 a.m., when she would often vomit food taken eight hours previously. There was no real pain, not more than a feeling of deep epigastric oppression, and no reference to the back or elsewhere. These attacks recurred about every two or three weeks. At the time of her seeking advice they were becoming more frequent and prolonged. She had received much treatment for them, sometimes with apparent relief, and she had gradually eliminated various articles from her diet under the impression that they did not agree with her, until she was now subsisting mainly on toast, dry biscuits, and milk.

Deformity of both hands had been present since birth. The patient had had measles, chicken-pox, scarlet fever, whooping-cough, and ' growing pains ' as a child. There had been no gastro-intestinal trouble until the present vomiting started, and the bowels had always opened regularly and continued to do so between the attacks. No history was obtained of any abnormality in the stools. Micturition was normal. The menopause occurred at 49 without any subjective symptoms. Recently there had been loss of weight. The patient's mother died of carcinoma ventriculi.

ON EXAMINATION.—The patient was a fairly well nourished, rather pale woman of short stature. The tongue was slightly furred but moist, the teeth were very good, and the fauces normal. Her heart was normal in size and situation. A systolic bruit was present at the mitral area conducted towards the axilla, and the pulmonary second sound was accentuated. Blood-pressure 154/84. The lungs were normal, and no abnormality was revealed in the abdomen on physical examination beyond a slightly flabby musculature and slight epigastric tenderness. Eyes and C.N.S. normal. The hands were small, and both showed imperfect development of the thumbs as well as short and atrophic-looking fourth and fifth digits. All the phalanges were present, and there was webbing between the left thumb and index finger. The urine was normal. Hb (Sahli), 87 per cent; blood-sugar (fasting), 100 mgrm. per 100 c.c.

X-ray Examination.—" The opaque food passed down the œsophagus and entered the stomach in a normal manner. The stomach is fairly normal in position. Tone good, peristalsis was normal along both curvatures. Gastric motility normal. There was no direct or indirect evidence of a gastric ulcer. There was no evidence of a permanent filling defect suggestive of a gastric neoplasm. The stomach was empty in two hours. The duodenal ' cap ' was well formed and showed no evidence of spasm or irregularity in outline suggestive

of a duodenal ulcer. In the middle portion of the descending part of the duodenum there was a persistent residue which was present at the end of forty-eight hours. This residue in the erect position appears to have a rounded base with a somewhat flattened upper margin.

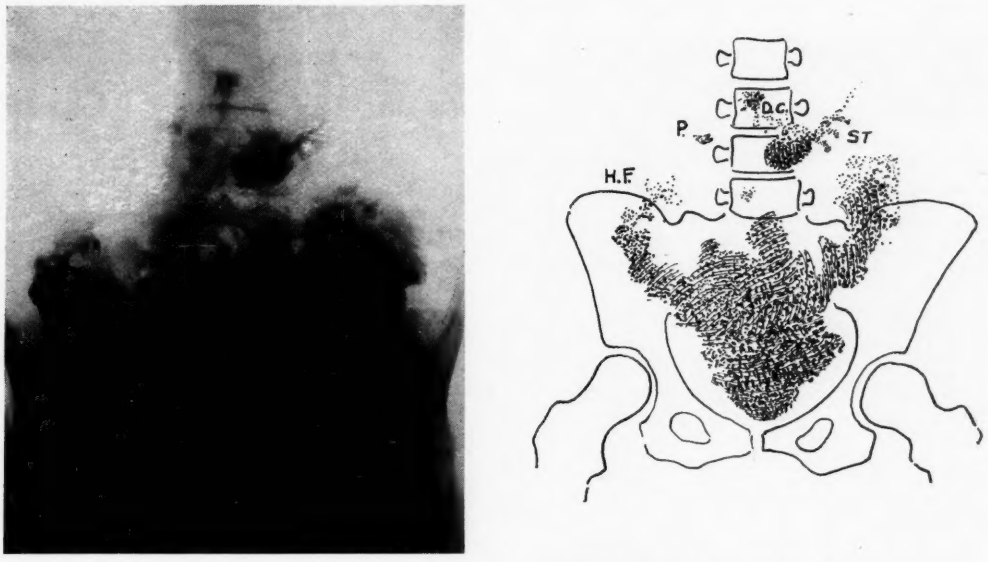

FIG. 554.—*Case* 2. Two hours after barium meal. P, Duodenal pouch; DC, Duodenal cap; St, Stomach; HF, Hepatic flexure.

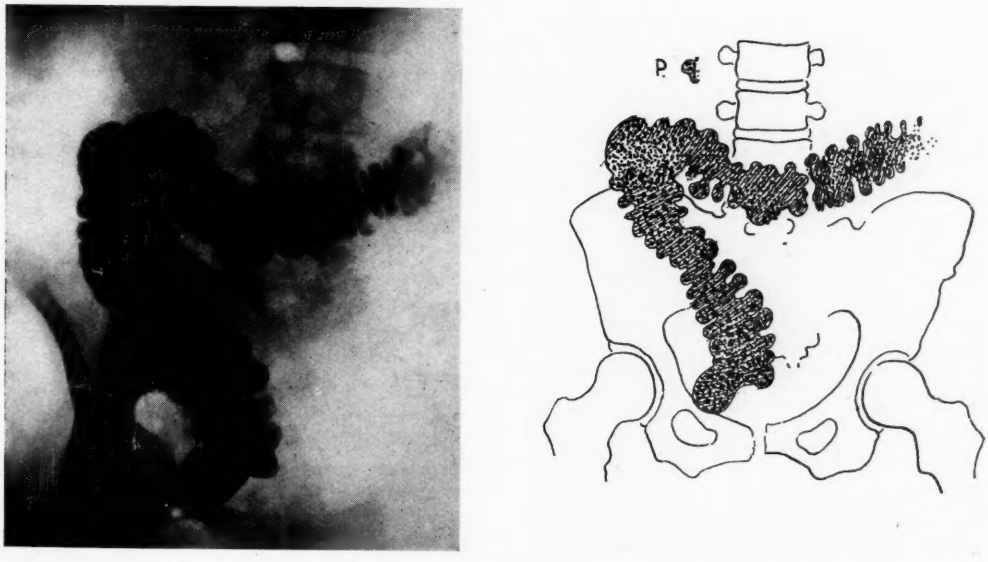

FIG. 555.—*Case* 2. Six hours after barium meal. P, Duodenal pouch.

The rounded base is suggestive of a small diverticulum. There are no indications of any gall-stones that are opaque to X rays. There is no definite evidence of any abnormality in connection with the large or small intestine." (*Figs.* 554–557.)

Fractional Test-meal.—This showed hypochlorhydria of mild degree together with slight delay in secretion, the highest figures for total acidity and free HCl being 35 c.c. and 10 c.c.

N/10 NaOH respectively, which was attained in one and a half hours. The form of both curves was normal.

OPERATION (H. N. F., Feb. 15, 1934).—Right paramesial incision. Stomach irritable with local patchy contractions, otherwise normal. Duodenum more mobile than usual. No

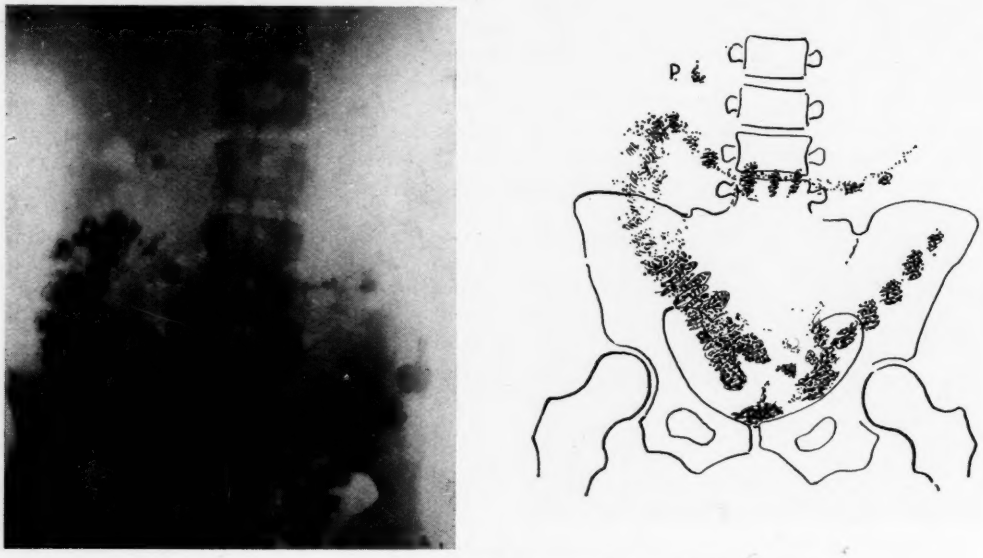

FIG. 556.—*Case* 2. Thirty hours after barium meal. P, Duodenal pouch.

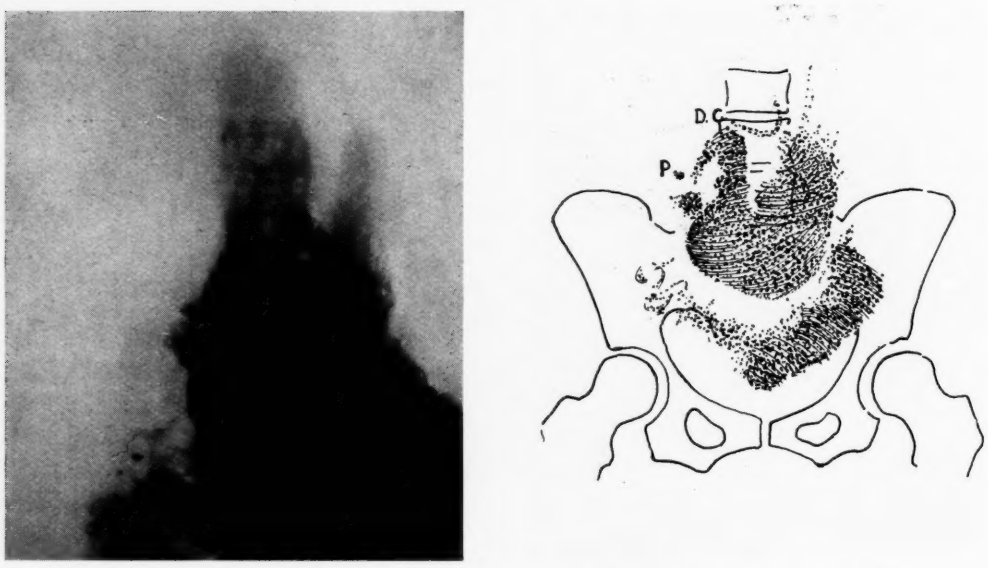

FIG. 557.—*Case* 2. Forty-eight hours after barium meal, with second barium feed just given. P, Duodenal pouch; DC, Duodenal cap.

outward abnormal appearances. Gall-bladder, slight subperitoneal deposit of fat; no adhesions or stones. Liver normal except for superficial fibrosis of capsule opposite rib margin. Ducts and pancreas normal. Colon, mild ptosis. Appendix free, no fæcoliths, fibrosis of terminal inch (left *in situ*). The duodenum was mobilized and turned over to the left.

Immediately the fundus of the diverticulum bulged on its outer side. The pouch, 2½ cm. long, was separated till its neck was defined, arising from the middle of the postero-internal aspect of the second part of the duodenum exactly where the branches of the pancreatico-duodenal arcade enter the bowel wall. Two small vessels, supplying the pouch on its superior and inferior surfaces, were ligatured. The neck of the pouch, ½ cm. in diameter, was divided between clamps, ligatured with catgut, and invaginated by two layers of fine catgut, over which the peritoneal incision was closed.

HISTOLOGY OF POUCH.—"In the sections the structure of the mucosa and submucosa is that of perfectly normal small bowel with abundant villi. Brunner's glands are absent. A few muscle fibres are present. There is no trace of inflammatory infiltration."

SUBSEQUENT PROGRESS.—The patient's convalescence from the operation was uneventful, and during the twenty-one months which have elapsed since then she has had no nausea or vomiting, and has been entirely free from all symptoms. It has been remarkable and most gratifying to see her delight in the re-discovery of the taste of foods "she dared not eat for years." "She no longer has to think about her diet but can eat anything", and has gained weight.

Case 3.—Miss D. E., aged 42, engaged in the Educational Department of the Sudan Government, consulted one of us (H. N. F.) when home on leave in July, 1929.

HISTORY.—For fifteen years she had been subject to bouts of epigastric pain and gastric flatulence which lasted three to four weeks and which had become worse and more frequent in the last two years. There was never complete digestive comfort between the attacks. An attack was usually ushered in by three or four days of diarrhœa, vomiting, and general abdominal distension. Then followed a month or so of deep-seated, gnawing epigastric pain, going through to the back but with no reference to the shoulders or elsewhere. It was pretty constant in intensity throughout the day. Lying down at night did not take it away, but she slept well. On waking she again experienced it. The pain had no definite relationship to food, though sometimes it was easier for half an hour or so after a meal. The quantity and quality of the food taken did not seem to influence the pain much, but she had little appetite and for the most part would confine herself to a light diet or 'slops' during an attack.

Eructation of wind was a marked feature, with some, but incomplete, relief, and hot water and sod. bicarb. helped in so far as they facilitated its expulsion. Nausea was occasionally present and, rarely, vomiting. The latter did not relieve the pain—indeed, it seemed to increase it. The bowels, always constipated, were kept active by 'purgen' or magnesia. Mucus in small quantity was now and then observed in the stools and there was always a good deal of intestinal flatulence. Menstruation was three-weekly, scanty, and without pain. Micturition was normal.

The previous history, beyond childish ailments and two short attacks of dysentery in the Sudan, revealed nothing important. Her work was strenuous and she had noticed that the attacks were apt to come on when she was overtired. All her teeth had been taken out two years previously, without any apparent benefit.

ON EXAMINATION.—When first seen in July, 1929, the patient was having a slight attack. The tongue was clean, her dentures were satisfactory, the tonsils and nasal passages apparently healthy. The thyroid, heart, lungs, and kidneys were all normal. The body was fairly well clothed. There was localized tenderness mid-way between the xiphisternum and umbilicus, no gastric or intestinal distension, and beyond a mobile and palpable right kidney nothing further of note in the abdomen. Per rectum the pelvic organs appeared normal.

X-ray Examination.—A barium meal and X rays showed some hypotonus of the stomach, which did not fully empty for five and a half hours. There was no evidence of ulcer in stomach or duodenum. A diverticulum of the duodenum was apparent in all films after the stomach had emptied, and some barium remained in it for twenty-four hours. Some general visceroptosis was present.

Diagnosis: The patient was of the highly-strung, extremely conscientious type. Though there was evident stasis in the duodenal pouch it seemed doubtful if this was the cause of her symptoms, which were regarded as being probably due to nervous overstrain in a sensitive subject, with, perhaps, a contribution from visceroptosis and constipation. She was therefore advised three weeks rest in bed, sensible feeding up, with bromide and belladonna and cod-liver oil and malt as medicine.

With this she improved and returned soon after to the Sudan. She was not seen again until her leave in the following summer. She had kept well for a few months after her rest but then the attacks returned.

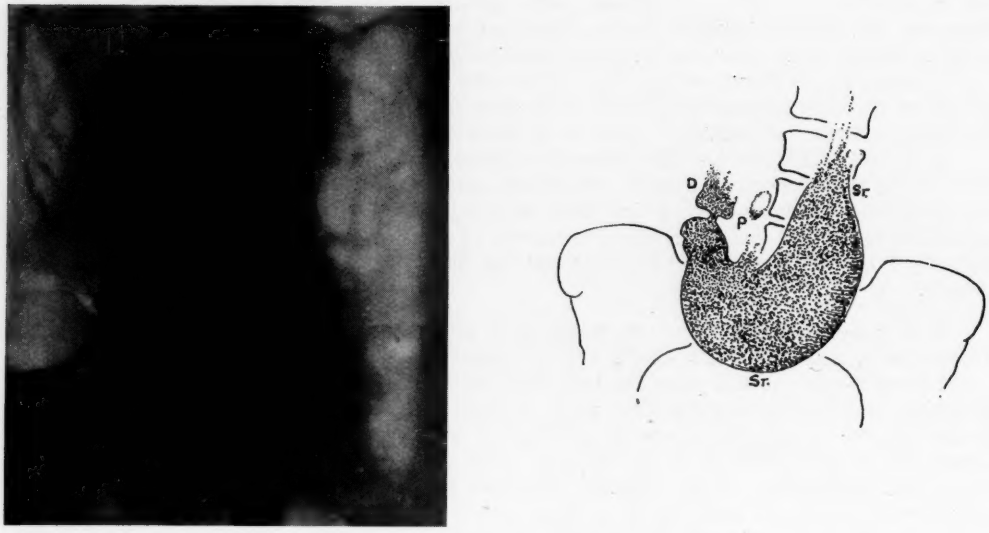

FIG. 558.—*Case* 3. Just after barium meal. St, Stomach; D, Duodenal cap; P, Pouch.

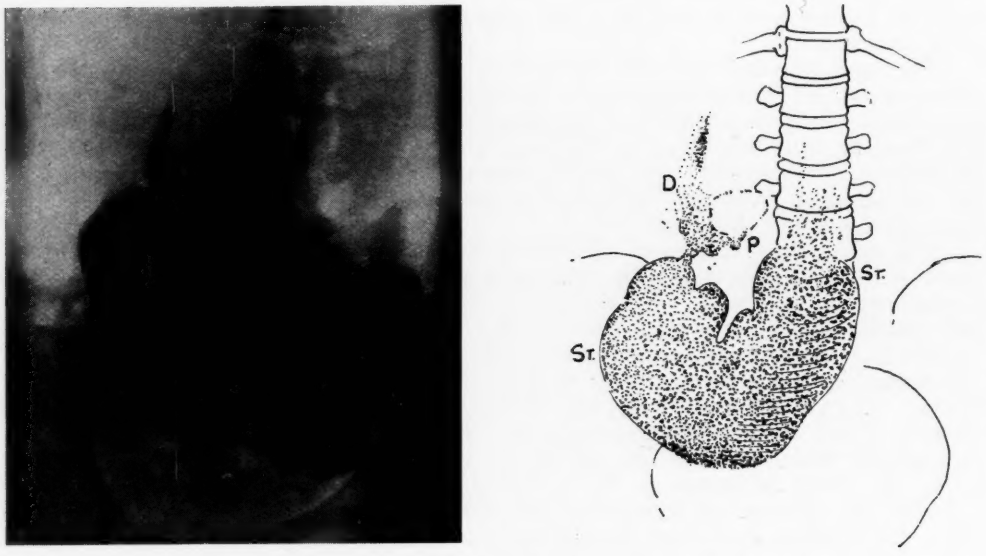

FIG. 559.—*Case* 3. Quarter of an hour after barium meal. St, Stomach; D, Duodenum; P, Pouch.

A further barium meal was given by Dr. Barrington Prowse in June, 1930, with the following report (*Figs.* 558–563). "Ptosis and displacement of stomach to the right—hypotonus but no delay in emptying—duodenal 'cap' in general well-formed but showed variable deficiency and want of tone on the inner side. In this region a diverticulum soon became evident communicating with the duodenum, though its stoma could not be seen. The shadow

was roughly triangular with the base downward and to the right, and it lay slightly above and to the inner side of the 'cap'. There was no delay in the duodenum itself, food passed through it normally, but a small quantity of bismuth persisted for many hours in the dependent corner

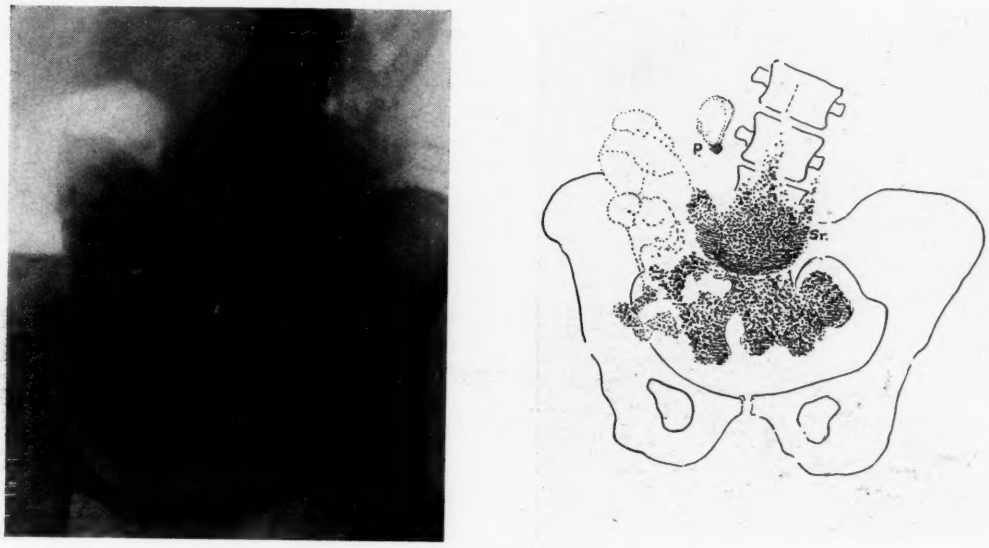

FIG. 560.—*Case* 3. Two hours after barium meal. St, Stomach; P, Pouch.

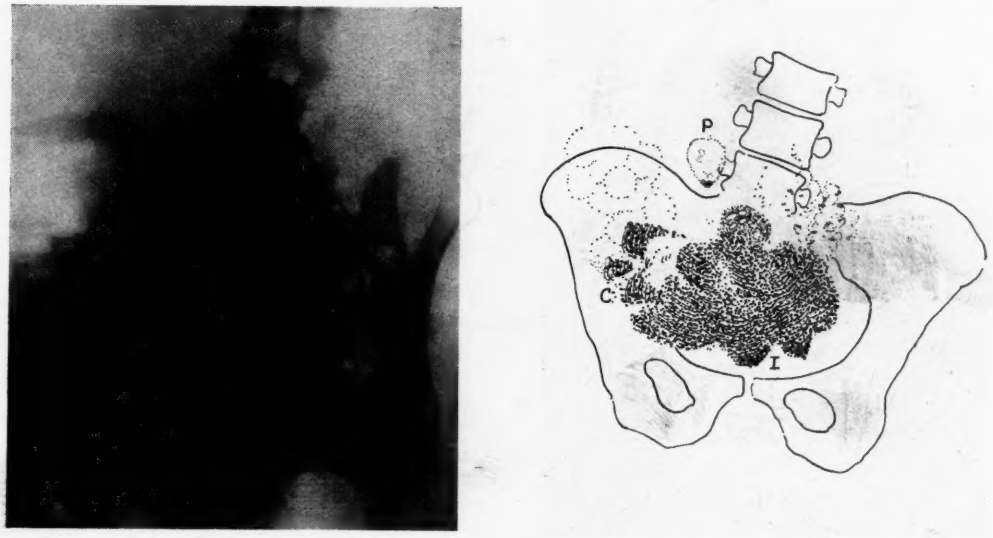

FIG. 561.—*Case* 3. Three and three-quarter hours after barium meal. P, Pouch; C, Cæcum; I, Ileum.

of the pouch. It could, however, be dislodged fairly easily in the supine position." Eight hours after ingestion the whole of the meal was in the colon. Shortly after this a second feed of barium emulsion was given and further observation made. It was found that the pyloric end of the stomach was closely applied to the hepatic flexure of the colon and could not be separated by palpation. There was a definite pressure tenderness in this region. "It seems

probable that adhesions exist and that they are the cause of the displacement of the stomach to the right and some drag upon the duodenal attachments. What relation they may have to the diverticulum it is difficult to say. There may have been a duodenal or gall-bladder

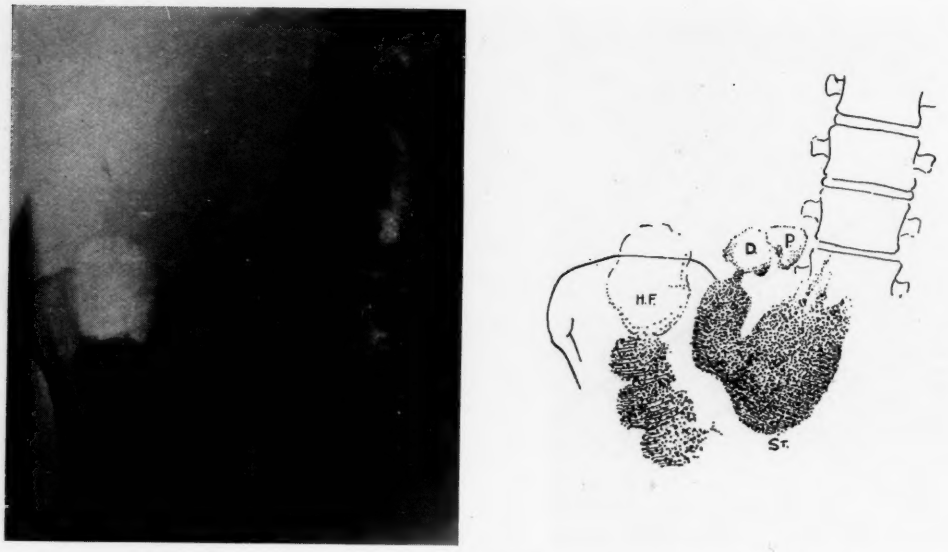

FIG. 562.—*Case* 3. Half an hour after second barium meal (upright position). St, Stomach; D, Duodenal cap; P, Pouch; HF, Hepatic flexure.

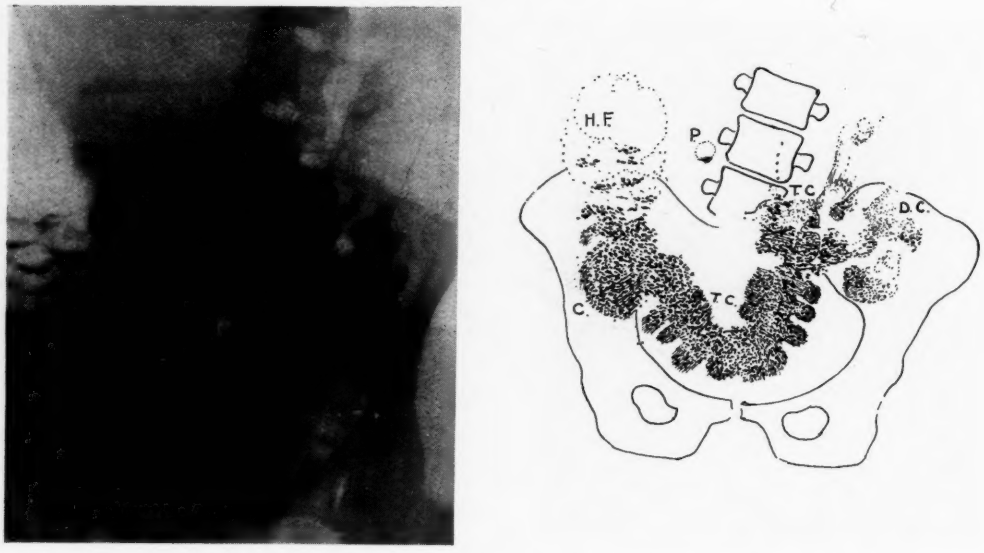

FIG. 563.—*Case* 3. Elght hours after barium meal. P, Pouch; C, Cæcum; HF, Hepatic flexure; TC, Transverse colon; DC, Descending colon.

lesion which has led to adhesions, or the whole thing may be secondary to some abnormality in development."

One felt that in view of this report an exploratory laparotomy was justifiable, but before doing this she was sent to Dr. John Ryle for his opinion. After a helpful analysis of her case, he concurred, and on July 2, 1930, a laparotomy was performed.

OPERATION.—The stomach was normal, no dilatation or hypertrophy. Just below the pylorus a rather well-marked thickening of peritoneum, probably congenital, and constituting a band ½ in. wide, crossed from the tip of the gall-bladder to the inner angle uniting the first and second parts of the duodenum. This, when divided, retracted sharply. There was no sign of gastric or duodenal ulcer. The gall-bladder was thin-walled, and half empty, contained no stones, and showed no signs of previous inflammation. There were no adhesions between the hepatic flexure and the gall-bladder or duodenum as suggested by the X-rays. The appendix looked normal and was left *in situ*. The proximal colon was not unduly mobile and there were no congenital membranes attached to it.

The duodenum was next examined, and anteriorly there were no signs of a diverticulum. It was therefore mobilized by incising the peritoneum on its right, turned over to the left until the small arcade of vessels entering the concave inner border was seen, and the pancreas came into view. Lightly adherent to the latter, flattened, and looking rather like one of its lobes, was the pouch, which was teased out of the connective tissue surrounding it. Its narrow neck, ½ cm. wide, was isolated, the two small vessels supplying it were divided and ligatured, a circular suture of fine catgut was placed around it before clamping, tying, and removing it, the circular suture tied, and a second layer placed over this. The pouch was empty and showed no signs of inflammation. Its neck entered the duodenum about halfway down the second portion, on the postero-medial border, practically in the same plane as the blood-vessels.

The common bile-duct was not seen, but its entrance into the duodenum was certainly below the diverticulum.

Microscopically the wall of the pouch showed muscle fibres at its proximal end, tailing off into mucous membrane and connective tissue at its fundus. There was no evidence of old inflammation, nor were there any Brunner's glands in the mucosa.

SUBSEQUENT PROGRESS.—The patient made an uneventful recovery. She returned to the Sudan in good health at the end of September, 1930. During the next three months the weather was hot and work very strenuous and when she was tired the pain and flatulence came on as before, though much less severely and for a few days only. There was no initial diarrhœa and vomiting, and the pain never went through to the back.

After the New Year 1931 she had a bad attack of cystitis. Since then she has had two minor attacks of the old pain lasting a week or ten days, both when overtired. But apart from these, appetite, digestion, and spirits have been excellent during the four and a half years that have elapsed, and in her own words "she has been an entirely different being".

Comments.—This case has been reported in detail in order that a judgement may be formed whether the marked change in her condition is to be regarded as due to the removal of the diverticulum or whether possibly the division of the band below the pylorus could have any bearing on it. It must be acknowledged that the latter may well have caused the displacement of the stomach to the right (as seen in the second X-ray examination) and possibly the want of tone in the duodenal 'cap'. But to our mind the syndrome of symptoms, particularly the initial diarrhœa and vomiting, described by others as sometimes associated with duodenal diverticula, seem more attributable to the presence of the pouch acting as a reflex source of irritation.

Though her whole health and outlook on life have been profoundly changed for the better, her case cannot be regarded as a 'cure' in the strict sense of the term.

As so often occurs in reflex disturbances, there would seem to have been two factors at work: (1) A directly determining one; (2) An underlying sensitive nervous system. The former removed, the latter was able to take care of itself, except in times of unusual stress.

SYMPTOMS

From these cases, and from the accounts given of others elsewhere, it would seem difficult to construct any definite syndrome of symptoms diagnostic of duodenal diverticulum when it causes trouble.

Attacks, i.e., periods of especial distress, occur, but there seems seldom to be complete digestive comfort between them, and the history may be a long one.

Pain, of a deep gnawing character, or a heavy discomfort in the epigastric region, is perhaps the most common symptom, and its relationship to food is somewhat vague.

Gastric flatulence and belching of wind, with incomplete relief of pain, was a marked feature in each of our cases.

Vomiting occurred in two cases, and in one it was so incessant as to constitute the outstanding symptom. It was noteworthy that in these two neither belching nor vomiting brought complete relief; indeed in *Case* 3 vomiting made the pain worse.

Diarrhœa always ushered in an attack in *Case* 3, and this symptom has been recorded by other observers.

Tenderness in the region of the diverticulum was present in some degree in all three cases.

As has been suggested, the demonstration of a duodenal diverticulum in a case in which prolonged and somewhat vague symptoms have been present, calls for the most careful search for other possible causes, and only when these have been eliminated, and treatment has failed, should operation be advised.

Our best thanks are due to Drs. Prowse and Cubbon for the help they gave us in X-ray investigations, and also to Dr. Galt for his reports on the histology of the specimens removed.

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FRACTURES OF THE NECK OF THE FEMUR

By R. WATSON-JONES, Liverpool

PATHOLOGICAL CONSIDERATIONS

Fractures of the neck of the femur conform to the same pathological laws as fractures of any other bone. It is now recognized that even in old people there is a free supply of blood to the femoral neck. Every surgeon who has operated on recent fractures has seen the capsule of the joint filled with blood, and the old theory that bone absorption and non-union were due to the inadequate blood-supply has been abandoned. The union of these fractures is governed by the rules of hyperæmic decalcification and ischæmic recalcification.[1, 2] When the fragments are imperfectly immobilized, the trauma of movement gives rise to continued

Fig. 564.—High fracture of femoral neck after nailing operation. Antero-posterior and lateral radiographs show that the fracture has firmly united without shortening of the neck.

hyperæmic decalcification. If there is no immobilization at all, this process goes on until the whole of the femoral neck has disappeared. If a plaster spica is used so that the fragments are fixed to some extent but without complete control of rotatory movement, there is a less severe degree of decalcification; nevertheless union is delayed, and if the fracture does unite, there is shortening of the neck. On the other hand, if by operative measures absolute immobility is secured, the fracture unites firmly[3] without decalcification and with minimal shortening of the bone (*Fig.* 564).

Although the fracture is freely supplied with blood, the greater part of this supply is derived from the distal fragment. The bone absorption following hyperæmic decalcification occurs mainly, therefore, in the distal fragment, and the head

remains practically unchanged. If the fracture is high enough to lie above all capsular attachments, the proximal fragment may be completely avascular, and in such a case there will be radiographic evidence several weeks later. The proximal fragment will retain its original density and will not participate in neighbouring decalcification[1, 4] (*Fig.* 565). The fact that a fracture of the femoral neck may reduce or even cut off the blood-supply of the proximal fragment has three important clinical applications.

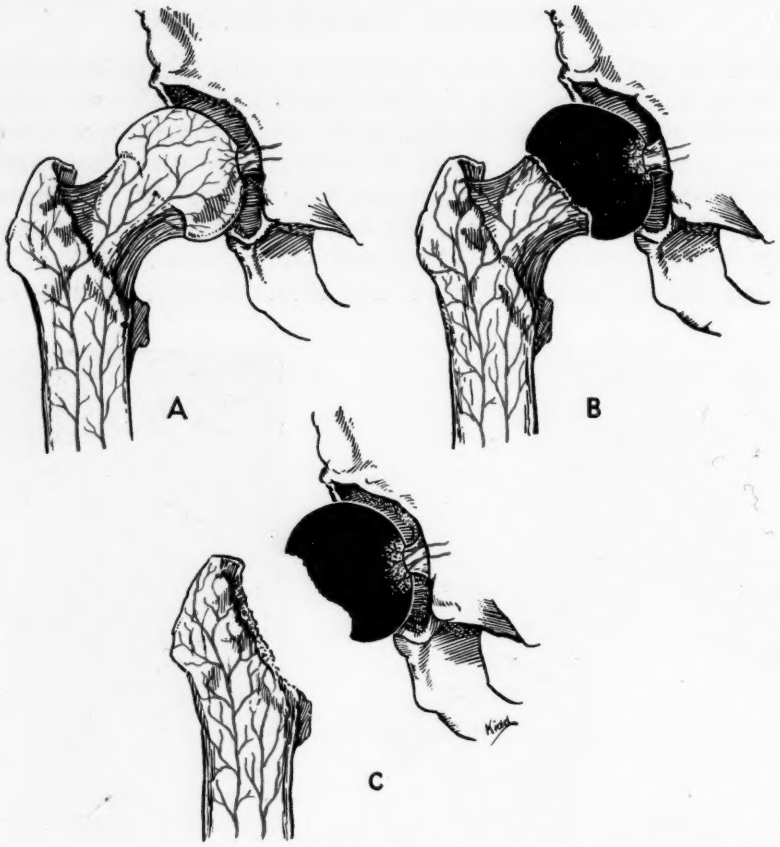

FIG. 565.—Blood-supply after femoral neck fractures. A, The head of the femur is supplied mainly by vessels running up from the neck; B, A subcapital fracture may almost completely deprive the head of its blood-supply; C, If the fracture is not immobilized the vascular neck undergoes hyperæmic decalcification and absorption. The avascular head retains its original density and shows neither decalcification nor absorption.

1. Slow Union of Subcapital Fractures.—In most fractures both the main fragments have an equally free blood-supply and both share in the development of a young connective tissue which will calcify to form callus. If one fragment is avascular, the whole of this new growth must develop from the other fragment, and union is correspondingly delayed. The union of high cervical and subcapital fractures of the femoral neck is therefore slow as compared with basal fractures. But although the vascular supply may account for slow union, it is not a cause of non-union. High fractures of the femoral neck will always unite, but only if immobility is more than usually perfect, and only if immobilization is continued

for four to six months or even longer. This is the main reason for the success of the Smith-Petersen nail.[5] For the first time in the history of the treatment of this injury a method has been devised which completely prevents rotatory as well as angulatory movement of the fragments, and which moreover allows immobilization to be continued for six or twelve months without hardship to the patient.

2. The Sequel of Osteo-arthritis.—It is well known that fractures of the neck of the femur are frequently followed by an arthritis of the hip-joint of the degenerative atrophic type (*see Fig.* 582). This arthritis is a sequel to the vascular disturbance which results from a fracture situated above all capsular attachments. The bone of the head of the femur can revascularize and regenerate by the invasion of new blood-vessels from the distal fragment. Articular cartilage is less tolerant, and if it is completely deprived of blood for any length of time it may undergo a degenerative change with resulting arthritis. It is obvious, therefore, that in every high femoral neck fracture absolute immobilization is a matter of urgency, not only to secure union of the fracture, but also to minimize the period during which the blood-supply of the articular cartilage is impaired.

This danger of arthritis of the hip-joint after subcapital fractures suggests that early weight-bearing is inadvisable in these cases. It seems probable that weight-bearing within a few weeks of operation, before the fracture is united and before the cartilage has regained its blood-supply, will increase the tendency to degenerative change.

3. Delayed Treatment and the Treatment of Ununited Fractures.—How long after the fracture has been sustained may the nailing operation still be employed? This depends on the extent to which the proximal fragment has lost its blood-supply, and the degree of decalcification which has occurred in the neck. In low fractures where the femoral head retains an almost normal blood-supply, the nailing operation may be practised many months after fracture, and the only contra-indication to operation is such a degree of absorption and shortening of the femoral neck that good movement is unlikely to be regained. This absorption cannot of course be estimated in the ordinary antero-posterior radiograph because the external rotation deformity gives an appearance of foreshortening of the neck. A lateral radiograph is essential.

On the other hand, the operation is inadvisable after the first six or eight weeks in high fractures where it is probable that the blood-supply of the head is impaired. Where the radiograph proves that the head is completely avascular the procedure is definitely contra-indicated. The degeneration of articular cartilage and osteo-arthritis which may develop even in cases operated on within a few days of injury will be inevitable if revascularization is still further delayed for several weeks or months. Moreover, the bone of the proximal fragment which has been avascular for several months has undergone necrosis and has become too fragile to withstand the pressure of the nail. In one such case a six-months-old subcapital fracture was nailed and the post-operative radiograph showed the nail perfectly placed in the middle of the femoral head. No weight-bearing was permitted and a plaster spica was applied for ten weeks. Nevertheless radiographs at two-monthly intervals showed the nail gradually ploughing its way upwards through the bone, until eight months later it lay in the joint between the head of the femur and the roof of the acetabulum.

Such cases should not be treated by any reconstructive nailing or bone-grafting procedure. They must be accepted as ununited fractures in which arthritis of the hip-joint is inevitable, and dealt with by means of a trochanteric bifurcation osteotomy.

TECHNIQUE OF OPERATION

Although there can be no doubt that a successful nailing operation is a most valuable procedure, an unsuccessful operation is disastrous. There are illustrations in the literature of penetration of the nail through the articular surface into the hip-joint. One such fracture has been seen where a surgeon's operation completely destroyed the articular surfaces and caused fibrous ankylosis. In other cases the nail has missed the proximal fragment altogether. Even apart from such gross misdirection there are other difficulties. In a subcapital fracture the greatest available depth of bone is little more than one inch, the greater part of which must be used if fixation is to be satisfactory. This is only available if the nail enters the head exactly in the middle of its fractured surface (*Fig.* 566). This is of much greater importance than that the axis of the nail should coincide with that of the neck. A nail which is accurately aligned but which lies marginally in the neck is only held in the head by a small sector of bone which may break off during the first few weeks and allow redisplacement of the fragments (*see Fig.* 575).

The Smith-Petersen Operation.—The technique originally described[5] consisted in exposure of the fracture from the front by Smith-Petersen's incision, with a second incision over the trochanter through which the nail was driven. The operation was associated with great difficulties. After a fairly extensive dissection it was still not easy to visualize the axis of the neck, and if the first attempt was unsuccessful it was almost impossible to extract the nail without damaging the bone. The fact that many modifications have been introduced to overcome these difficulties does not detract from, but rather pays tribute to, the genius of the inventor of the nail.

Directing Guides.—The necessity for some mechanical means of directing it became apparent at an early stage. Several types of directing apparatus were made by instrument makers in Liverpool, but all of them were discarded sooner or later because they complicated the technique and were not always reliable. Various forms of guide and aiming apparatus have also been devised by Delbet, Sven Johansson,[6] Lloyd,[7] and Stirling.[8]

Cannulation of the Nail.—The preliminary insertion in the bone of a wire drill in order to guide a cannulated screw was described by Max Richard[9] and by Henschen[10] even before Smith-Petersen described his nail. The application of this principle to the three-flanged nail occurred independently to several surgeons. Sven Johansson,[6] of Gothenburg, and King,[11] of Melbourne, both used centrally cannulated nails and Kirschner wire, and both have developed an extra-articular technique relying on radiographic control. The author's nails[12] were constructed with a wider cannula to take a much heavier guide which would not easily bend or displace, and the technique was applied to the open operation so that radiographic control was not essential. Two types of nail were devised, one with a central cannula and the other cannulated along the margin of one of the blades. If, when the guide had been driven in, it was found to lie centrally in the femoral neck,

a centrally cannulated nail was used; if the guide was found to be eccentric, a laterally cannulated nail was chosen. In practice it was found fairly easy to insert the guide accurately, and the laterally cannulated nail was ultimately discarded.

Stainless Steel Nails.—The first nails produced in England were plated or made of ordinary steel. It was found that these nails gave rise to a severe local

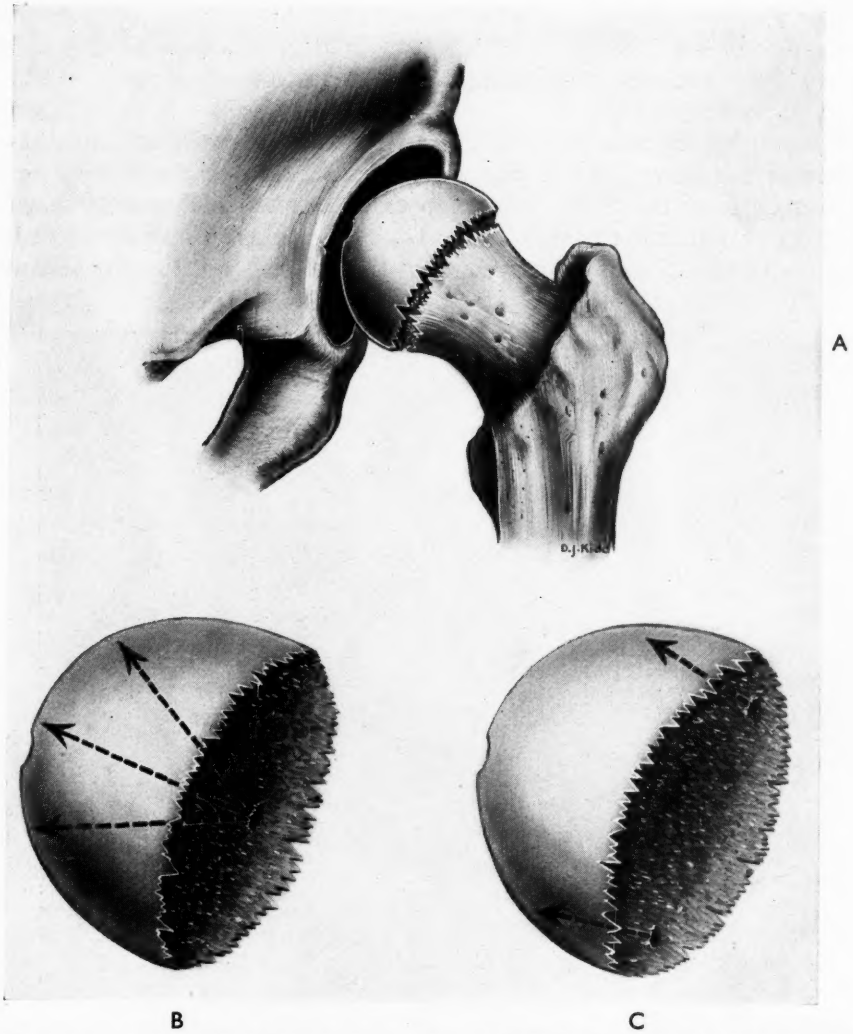

FIG. 566.—A, In a subcapital fracture the greatest available depth of bone may be little more than 1 in.; B, This depth of bone is only available if the nail enters the head exactly in the middle; C, Even if a nail is accurately aligned, if it enters the margin of the head it is only held in position by a thin sector of bone which may break off and allow redisplacement.

reaction with decalcification of the bone and loosening of the nail. When stainless steel was employed this complication did not arise.[3]

Exposure of the Fracture.—Smith-Petersen's anterior incision with a second small incision over the trochanter is associated with all of the difficulties of blind insertion. Apart from confirming the accuracy of reduction, the only value of the extensive dissection lay in recognizing mal-alignment of the nail when it was too

late. The incision was therefore modified by curving its lower end backwards, raising a flap, and so exposing the femoral neck from the fracture to the trochanter. The operation was very much facilitated, but in one old and feeble patient with impairment of circulation the whole flap sloughed and the patient died after ten weeks.

FIG. 567.—X-ray measuring rod, graduated in quarters of an inch.

A lateral approach is now used, and it is found that through a 6-in. incision the head of the femur, the fracture, the whole length of the femoral neck, and the upper shaft of the femur can be so easily exposed in one field of view that it is a very easy matter to aim accurately. This change in operative exposure has done as much to facilitate the procedure as the use of a guide and cannulated nail.

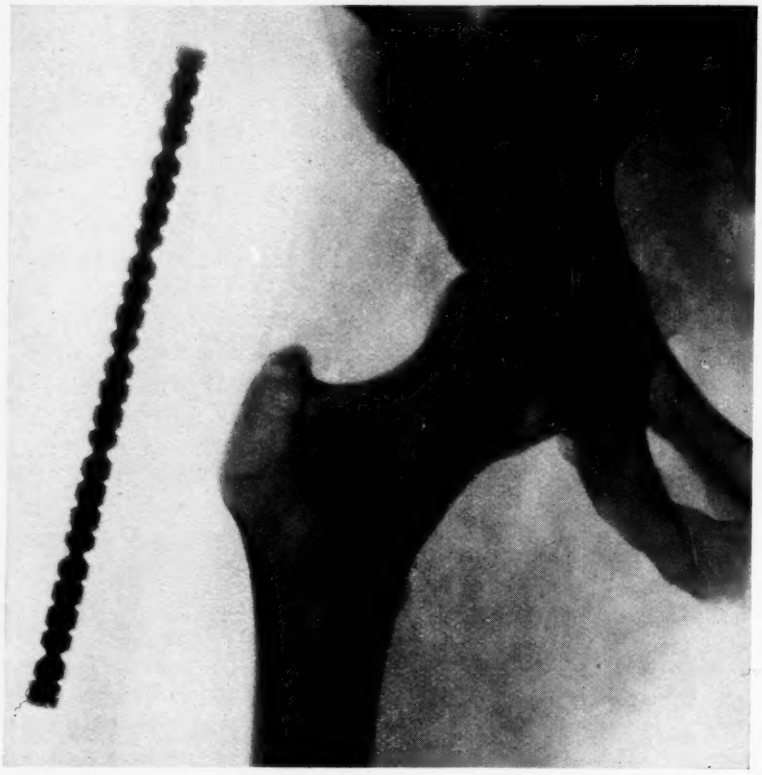

FIG. 568.—The rod is strapped on to the normal thigh at the level of the femoral neck. A film is taken with the hip abducted and internally rotated. The length of the nail required is determined by measuring the shadow of the bone against the equally magnified shadow of the rod.

The Author's Operation.[12]—

Length of Nail.—The correct length of nail is determined with absolute accuracy before the operation is begun. The surgeon must be sure that the nail is long enough to engage with the greatest possible depth of bone in the proximal

fragment. Equally he must be confident that the nail can be hammered home without penetrating the joint. If the head of the nail is not firmly punched into the cortex, the nail may slide out of the proximal fragment before the fracture is united (*see Figs.* 578, 579).

The X-ray shadow, being magnified, cannot be measured directly. Moreover, the degree of magnification depends on the distance of the tube from the limb, and standardized calculations are therefore fallacious. A measuring rod graduated with $\frac{1}{4}$-in. grooves has been made (*Fig.* 567). This is strapped on to the outer aspect of the thigh level with the neck of the femur on the uninjured side; the hip is fully abducted and internally rotated and a film is taken (*Fig.* 568). The true length of the bone may now be determined by measuring the shadow of the femoral neck against the equally magnified shadow of the grooved rod.

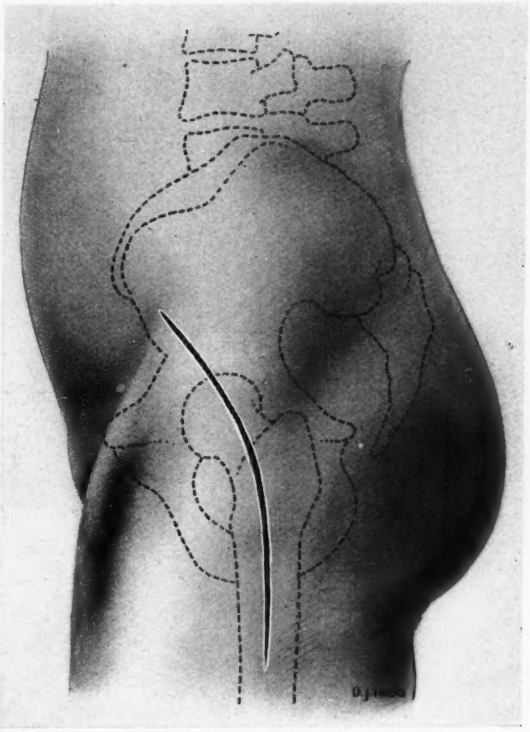

Fig. 569.—Lateral approach for open reduction.

Operative Exposure.—Spinal anæsthesia is used except in cases of low blood-pressure. The neck of the femur is exposed in the interval between the gluteus medius and the tensor fasciæ femoris (*Figs.* 569, 570). To assist in retracting the gluteus medius sufficiently to see the side as well as the front of the neck, the anterior fibres of insertion are dissected off the trochanter. If a more free exposure is required an osteotome is driven into the front of the trochanter, which is levered out on its intact posterior margin (*Fig.* 571). Separation of the trochanter, however, involves a good deal of hæmorrhage, whereas if this is avoided the operative field is almost bloodless. The capsule is incised along the upper border of the neck, dissected away from the intertrochanteric line, and turned forwards as a triangular flap. If the upper fibres of the vastus externus are now turned down subperiosteally, a wonderfully clear view of the whole line of the neck is secured.

Insertion of Guide and Reduction of Fracture.—The point of entrance of the guide is chosen exactly in the middle of the lateral surface of the femur $\frac{1}{2}$ to $\frac{3}{4}$ in. below the trochanter, and the cortex is perforated with a gouge to allow the guide to be driven in easily. The guide is bored in by means of a simple T-shaped handle (*Figs.* 572, 573) and is inserted and re-inserted as frequently as necessary until it is seen to emerge from the exact centre of the fractured surface. The fracture is now reduced accurately and the guide is driven into the proximal fragment. Since it is known that the guide lies in the middle of the distal fragment, it

is only necessary to secure perfect reduction of the fracture to be certain that the guide will enter the middle of the head. Perfect reduction of the fracture is also of great importance because the final range of hip movement depends entirely on the accuracy of reduction. If the fragments are too much impacted, the neck will be shortened and abduction movement limited. If there is any rotational displacement, flexion movement will be limited exactly as it is in epiphysial coxa vara. *Fig.* 574 illustrates the range of joint movement in a typical series of cases. Comparison of these photographs with the lateral radiographs shows that in every case

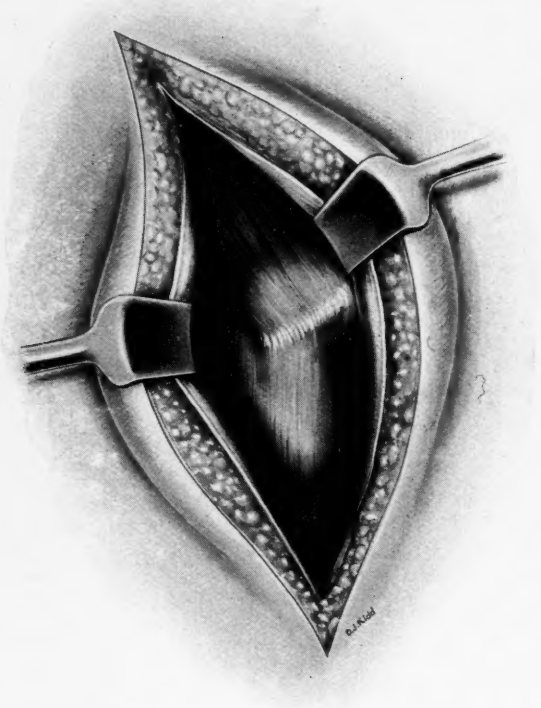

FIG. 570.—The anterior border of the gluteus medius is defined and retracted outwards. The upper fibres of the vastus externus are turned down subperiosteally.

where hip flexion is limited there is some degree of rotational displacement, whereas in every case where hip movement is normal the fracture has been perfectly reduced.

The guide is of sufficiently heavy material to steady the fragments and to prevent re-displacement while the nail is being inserted. The Smith-Petersen retractor, which is only insinuated with great difficulty between the articular surfaces of the joint, becomes unnecessary, and since it is an instrument which must aggravate the tendency to arthritis it should be discarded.

Insertion of Nail.—The nail is threaded over the guide and hammered in with a cannulated punch. It is important to see that the guide engages accurately with the cannula of the punch and that it is not driven still further into the bone as the nail is hammered home (*see* p. 801). If the fragments have separated slightly as the nail entered the proximal fragment, the gap is closed by the Smith-Petersen

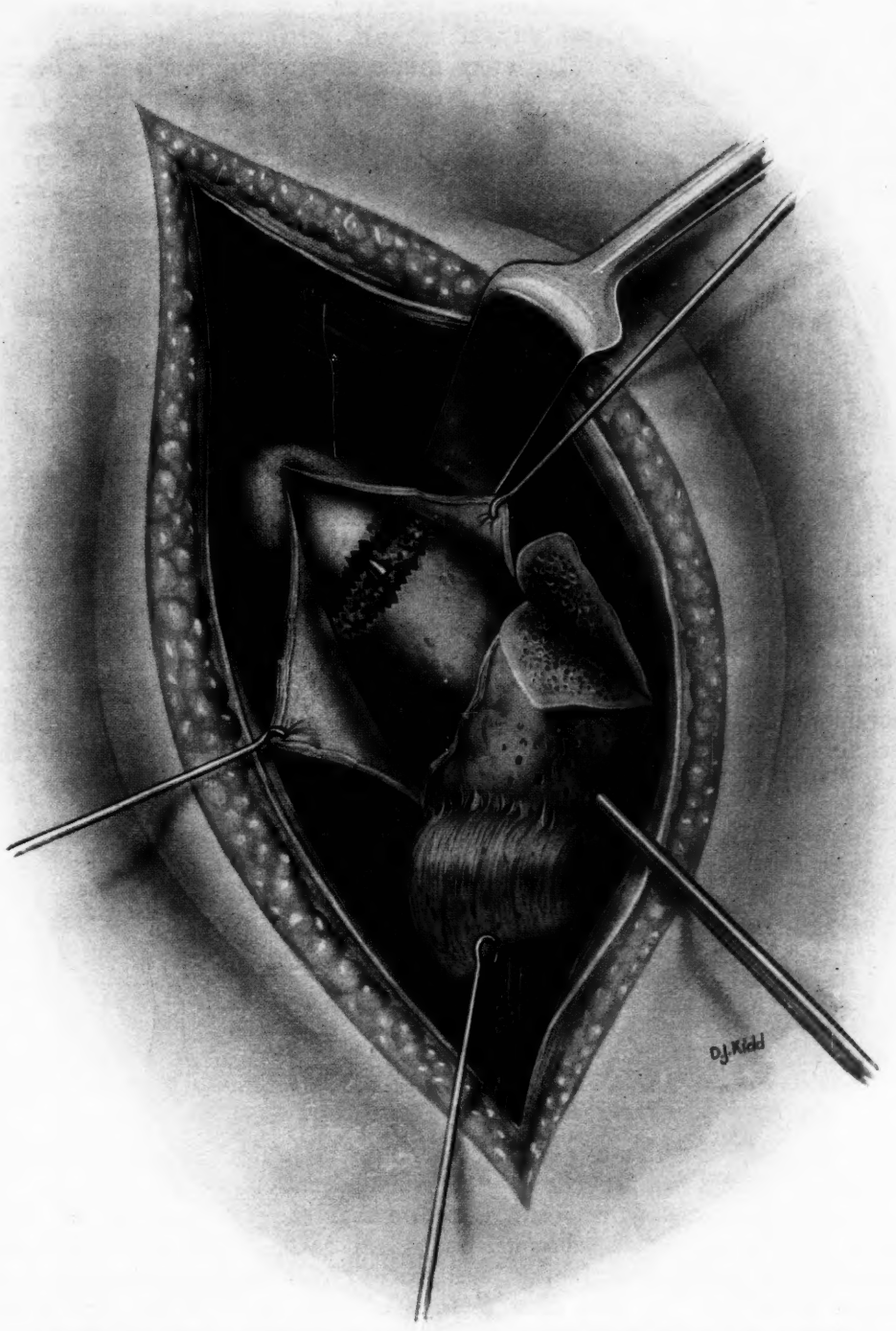

FIG. 571.—For a more free exposure the front of the trochanter is levered out on its intact posterior margin. The capsule is turned down as a triangular flap to expose the whole length of the femoral neck. The point of the guide must be seen exactly in the middle of the fractured surface before the fracture is reduced.

impaction punch. This punch should be used very cautiously, and no more should be done than to close the gap. Belief in the importance of impaction is a relic of the days when there was no other means of arresting rotational movement of the fragments. Now that this movement is controlled by the three-flanged nail, these fractures unite just as certainly whether they are impacted or not. Blind and uncontrolled impaction is not only valueless, but it destroys the cancellous bone, shortens the femoral neck, and may increase the tendency to

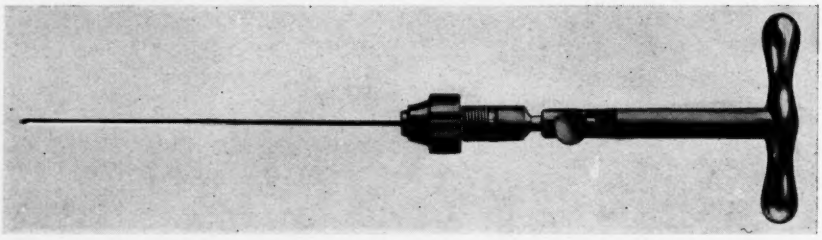

FIG. 572.—The directing guide is held in a chuck which will fit either a T-shaped handle or a brace. Direction can be more accurately controlled if the guide is bored in with the T handle; but if the bone is unduly hard, the guide is drilled in with a brace.

arthritis by bruising the articular surfaces. Cautious use of the impaction punch is specially important if closed methods are used and the fracture is not visualized.

After-treatment.—No plaster spica or other external fixation is used. A Thomas's splint is dangerous because it converts the limb into a long rigid lever which transmits every movement exactly to the level of fracture. Moreover, knee movement must begin the day after operation. Some simple measure should be used to prevent the limb from turning into the externally rotated position. A shoe

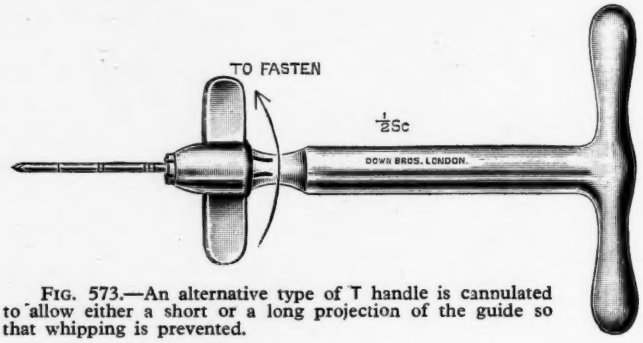

FIG. 573.—An alternative type of T handle is cannulated to allow either a short or a long projection of the guide so that whipping is prevented.

or bedroom slipper with a 6-in. piece of wood nailed on to the heel and projecting outwards so that it lies flat on the bed is quite adequate.

In low transcervical fractures it is safe to begin weight-bearing in four to six weeks (*Fig.* 575). In high subcapital fractures there should be no weight-bearing during the first three months nor until there is radiographic evidence that the fracture is uniting. In most cases there is evidence of union in three or four months and of final consolidation in four to six months. Even though weight-bearing is not allowed, the patient sits up and turns regularly in bed. No more than this is necessary to avoid hypostatic congestion even in the oldest of patients, and early weight-bearing is not imperative.

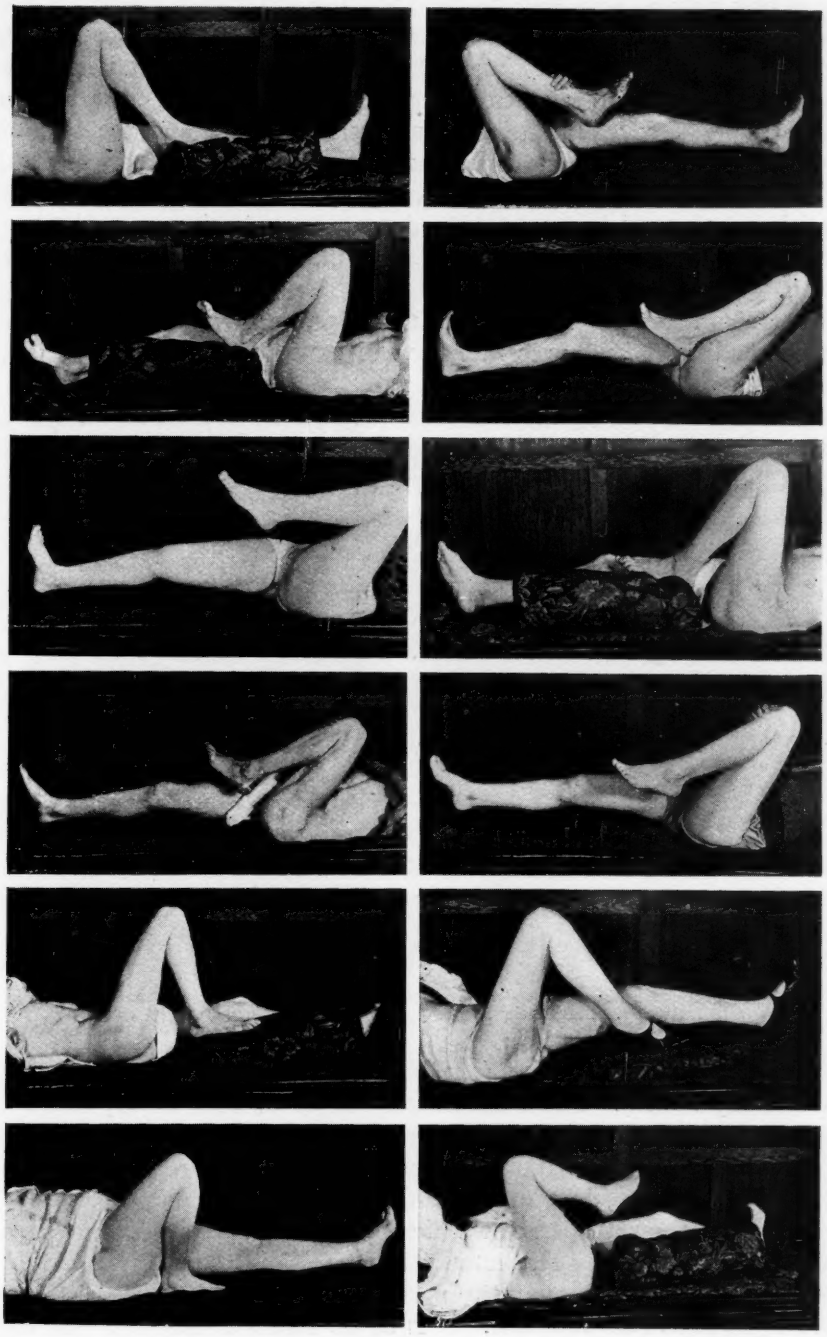

FIG. 574.—A typical series of end-results showing the best and the worst ranges of hip and knee movement. Limited hip flexion is always due to inaccurate reduction of the fracture (rotational displacement).

The Extra-articular Operation of Sven Johansson[6] and King.[11]—The fracture is first reduced on a traction table and the accuracy of reduction confirmed by antero-posterior and lateral radiographs. A 3- or 4-in. incision is used over the great trochanter and one or more Kirschner wires are drilled into the neck. Sven Johansson relies on mathematical calculations and an elaborate directing apparatus. King uses several wires, the first inserted behind the femoral neck under fluoroscopic control, and the others in the bone. Whatever devices are used to assist in directing the wires, antero-posterior and lateral radiographic control is essential and the nail is not introduced until one wire is seen to be in perfect position. Radiographs are again taken after the nail is inserted and the wire withdrawn.

Comparison of the Blind and the Open Operations.—Similar extra-articular methods have been used in a few patients who were thought to be too old

FIG. 575.—In transcervical fractures with an accurately placed nail weight-bearing may begin in four to six weeks. Early weight-bearing is not permitted in subcapital fractures.

and feeble for the open operation (one being an old woman of 91 whose fracture was successfully nailed). Local anæsthesia was used, with a very short incision just long enough to admit the nail and punch. No directing apparatus was employed but there was radiographic control throughout all stages. The procedure is a long and tedious one, the operative time varying from one to two and a half hours, whereas the open operation which has been described can be completed in thirty to forty minutes. The extra-articular method can only be carried out if a traction table, one or preferably two portable X-ray machines, and excellent team work and organization are available. It is by no means certain that this technique causes less shock than the open operation. With the 'open' procedure there is a 6-in. incision, rather more hæmorrhage, and a rapid operation; with the 'closed' technique a 2- to 4-in. incision, rather less hæmorrhage, and a prolonged operation. The one advantage of the blind method over the open one is the reduced risk of

sepsis, and King freely admits that this is the main reason for his advocacy of the extra-articular operation.

The position may therefore be summarized by saying on the one hand that no surgeon should attempt the extra-articular or blind operation unless he can command expert team work and radiographic control in the operating-theatre, and on the other hand, that no surgeon should attempt the open operation unless he can be quite confident of asepsis, and of a high standard of operative skill and judgement.

BASAL FRACTURES OF THE FEMORAL NECK

Since Whitman's work[13] was first published it has been possible to rely on good bony union of basal fractures of the neck of the femur treated in a plaster spica. For this reason the nailing operation has not been generally applied to fractures at this level. Nevertheless the plaster spica is associated with considerable difficulties and discomforts. Severe stiffness of the knee frequently develops, and unless the plaster is retained for four to six months coxa vara and shortening usually appear. Experience with high fractures has shown that the post-operative convalescence is so much more comfortable and so relatively free from complications, that during the last eighteen months we have been nailing basal as well as cervical fractures wherever possible.

Even greater difficulties may be experienced than in the nailing of high fractures. One of the first cases failed because the nail was inserted in the usual position ½ in. below the lower margin of the great trochanter. At this level the bone of the distal fragment is usually too thin to secure a firm grip of the nail. Although the nail becomes firmly embedded in the proximal fragment it is still possible to move the distal fragment on the nail and the fracture is not completely immobilized. In later cases the nail has been driven in at a much lower level, usually 1½ to 2½ in. below the trochanter, so that it lies more obliquely and is buried in not less than 1 in. thickness of bone below the fracture (*see Fig.* 578). The shaft of the femur, however, is much more prone to splitting than the trochanter and neck, and the nail must be hammered in with great care. The tendency is to split upwards from the nail to the fracture, so that none of the three blades of the nail should point in this direction. Before the guide or nail is inserted a hole should be gouged out of the cortex only slightly less than the diameter of the nail. If a split does appear in the cortex its extension upwards must be prevented by making a small cross-cut with a fine osteotome or saw. Before embarking on the operative fixation of pertrochanteric and intertrochanteric fractures, lateral as well as antero-posterior radiographs must be carefully examined in order to be sure that the comminution of the upper femoral shaft will not prove an insuperable obstacle.

PATHOLOGICAL FRACTURES OF THE NECK OF THE FEMUR

An attempt was made in one case to nail a fracture of the neck of the femur in a case of osteitis deformans. In Paget's disease fractures of the femoral neck show the same interference with the blood-supply of the head, the same delay in union and tendency to non-union which is observed in uncomplicated cases. The

operation was undertaken with some reluctance, but since the bone had fractured and not bent, it was thought that the disease must have advanced beyond the stage of softening to the stage of recalcification. It seemed possible that a nail might succeed in immobilizing the fracture and overcoming the joint stiffness, which is particularly difficult in cases of Paget's disease immobilized in plaster. The operation was most difficult owing to the thickening and vascularity of the bone, and, moreover, with the last punch of the hammer the bone split down the whole length of the neck. The nail was withdrawn and a double plaster spica applied. The fracture did ultimately unite after six months of immobilization. Experience in this case discourages any further attempt to apply the nailing method to femoral neck fractures in Paget's disease.

In one other case there was a pathological condition of the bone which was not recognized before operation. The patient had previously sustained a fracture of the shaft of the humerus from a trivial injury, and the X-ray showed undue sclerosis of the neck of the femur but no evidence of a generalized skeletal disorder. When the fracture was exposed it was found quite impossible to drive the guide beyond the cancellous tissue of the trochanter into the neck, the bone of the neck being extraordinarily dense and hard. An attempt was made to drill the bone but this also failed. The nail was ultimately inserted with very great difficulty after a track had been cut with a narrow gouge.

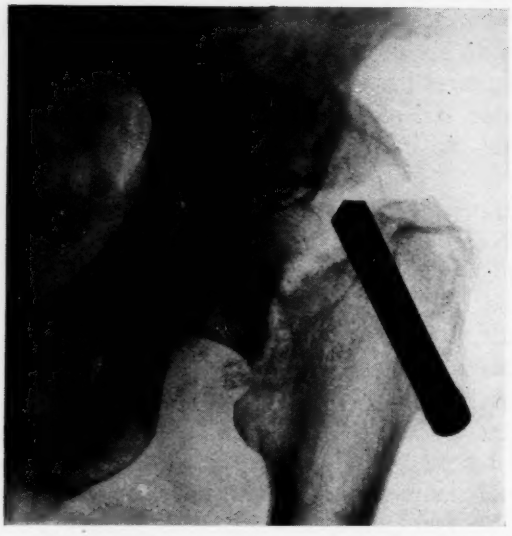

FIG. 576.—Re-fracture three weeks after an apparently successful operation, with re-displacement and 'foreshortening' of the neck due to external rotation. The nail though in good alignment lay marginally. A marginal fragment of bone has broken off the proximal fragment and lies above the point of the nail. The complication may now be avoided by the use of a guide and cannulated nail.

Twelve months later the lower half of the fracture is united, but there is decalcification and absorptive change in the upper half. It is proposed to remove the nail and apply a plaster spica for two to three months in the hope that consolidation will be completed. No evidence is yet available as to the underlying cause of the osteosclerosis.

COMPLICATIONS

Re-fracture due to Inaccurate Insertion of Nail.—This complication was only observed in the earlier cases of the series, but at that time it represented one of the great difficulties of the operation (*Fig.* 576). The use of a preliminary guide and the new exposure of the fracture has solved this problem. The introduction of Smith-Petersen nails without the use of a preliminary guide, and without either radiographic control or operative exposure of the fragments, should no longer be countenanced.

Wound Infection.—There was only one case in this series of 64 operations where there was any infection of the wound. The infection was of a low grade, but the formation of a sinus necessitated removal of the nail. The open operation should not be performed unless asepsis can be relied upon, and no-touch technique must be adopted exactly as in any other joint operation.

There was one other case, already referred to, of aseptic necrosis of the flap in a uræmic patient with auricular fibrillation. Since all of these patients are old and many have circulatory failure, flap incisions should be avoided.

FIG. 577.—Basal fracture after nailing. The guide was inadvertently driven in with the nail. Extraction of the guide was made still more difficult by its breaking in the middle of the femoral head just beyond the point of the nail.

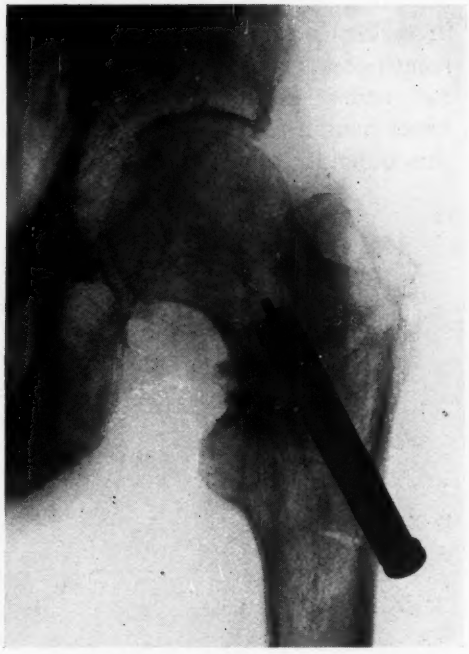

FIG. 578.—Same case as *Fig.* 577 after intra-abdominal extraperitoneal removal of the broken fragment of guide three weeks after first operation. In basal fractures the nail is driven into the shaft at a lower level, in order to gain a firm grip of the distal fragment. The nail is beginning to slide out of the bone.

Penetration of Guide into Acetabulum and Pelvis.—In one case there was an unexpected complication which gave rise to serious difficulty. When the guide is in position in the femoral neck, the length of guide projecting beyond the bone may be less than the length of the nail, so that when the nail is threaded over it the guide is obscured. In this case the nail was punched home without first confirming the fact that the guide was threading satisfactorily through the cannula of the punch. Only when the nail was completely embedded was it discovered that the guide had gone right through the hip-joint and pelvis. It was impossible to extract it without extracting the nail. Radiographs showed that the situation was still more difficult, for the guide had fractured in the middle of the femoral head just beyond the point of the nail (*Fig.* 577). The problem was to extract a 4-in. fragment of metal with one end in the middle of the femoral head and the other end in front of the sacro-iliac joint. It was successfully removed

three weeks later by an incision along the crest of the ilium and Poupart's ligament, stripping up the periosteum of the ilium, the iliacus and the peritoneum, localizing the guide in the true pelvis, cutting it in half, and extracting each half independently (*Fig.* 578). The patient recovered with no sign of damage to the hip-joint.

Now that this complication is recognized it is unlikely to recur. It is an easy matter when the nail is half embedded to be satisfied that the guide is passing smoothly through the cannula of the nail and punch.

Penetration of Nail into Hip-joint.—The use of too long a nail so that the point emerges through the articular surface of the femur was a constant fear in earlier cases. Several examples have been recorded. Now that the necessary length of the nail is calculated before operation, using the special graduated X-ray rod, and measuring the distance in the normal hip from a point $\frac{3}{4}$ in. below the lower margin of the great trochanter to a point $\frac{1}{4}$ to $\frac{1}{2}$ in. from the articular surface, this difficulty does not arise. In older cases where there has been absorption of the bone this measurement of the normal hip may be fallacious. In such a case the depth of the proximal fragment should be estimated before operation by the measuring rod, and the depth of the distal fragment measured during the operation. When the point of the guide has just appeared at the fractured surface the length of guide still projecting beyond the bone is measured and subtracted from the total length of the guide already known.

FIG. 579.—Subcapital fracture three and a half months after nailing. Originally the nail was well embedded in the proximal fragment, but it has slid down its groove in the bone, leaving a small fragment of metal behind. Fortunately, the fracture had already united.

Loosening of Nail from Bone Absorption.—It might be thought probable that absorption of bone round the nail due to an irritative hyperæmic decalcification would account for frequent failure. Decalcification of such a degree as to endanger fixation of the fragments was only observed in the earliest cases when plated nails were used. With stainless steel nails there is very much less reaction. That there is some decalcification is undoubted, and the remarkable tightness of the nail when it is first driven in is not maintained for more than a few weeks.* After that time it is not difficult to slide

* The fact that in post-mortem specimens the nail after the lapse of many months may still be so firmly gripped by the bone that it cannot be moved may prove misleading. The shrinkage of the adjacent bone as the post-mortem specimen dries is sufficient to grip the nail once more, and it does not follow that the nail was equally tight during the life of the patient.

the nail up and down its socket in the bone; but the genius of the invention lies in the fact that a three-flanged nail still lies in a three-grooved socket, so that rotational movement is still impossible and there is no interference with the consolidation of the fracture.

FIG. 580.—Stainless steel domed cap which is screwed over the head of any nail which shows a tendency to slip out of the bone.

In many cases this longitudinal movement of the nail is of such a degree that the head of the nail becomes more and more prominent as it slides out of the bone, and tenderness over the nail head is one of the definite indications for removal

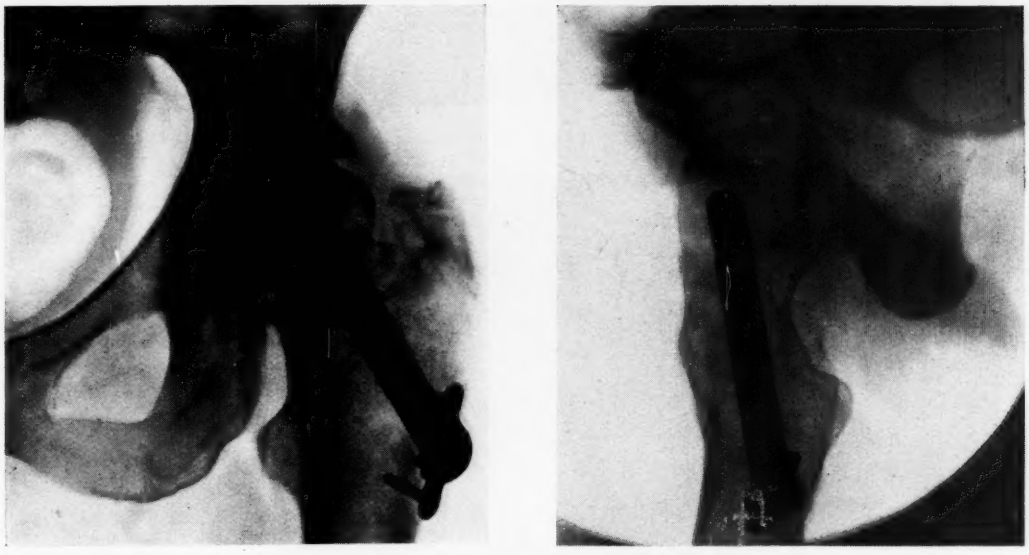

FIG. 581.—Cervical fracture showing the domed cap in position over a cannulated nail. The cap is not applied as a routine, but only after six or eight weeks if there is evidence of displacement of the nail.

of the nail. This seldom occurs in less than six to twelve months, but in one case where the nail head had not been firmly punched into the cortex of the femur the nail slid down so rapidly that its point was disengaged from the proximal fragment as early as three months after operation (*Fig.* 579). Fortunately repair was rapid in this particular case and the fracture had already united. To meet this difficulty a stainless steel cap has been made (*Fig.* 580) which can be screwed over the head of the nail. It is not used as a routine, but is reserved for any case in which longitudinal displacement develops six or eight weeks after operation (*Fig.* 581).

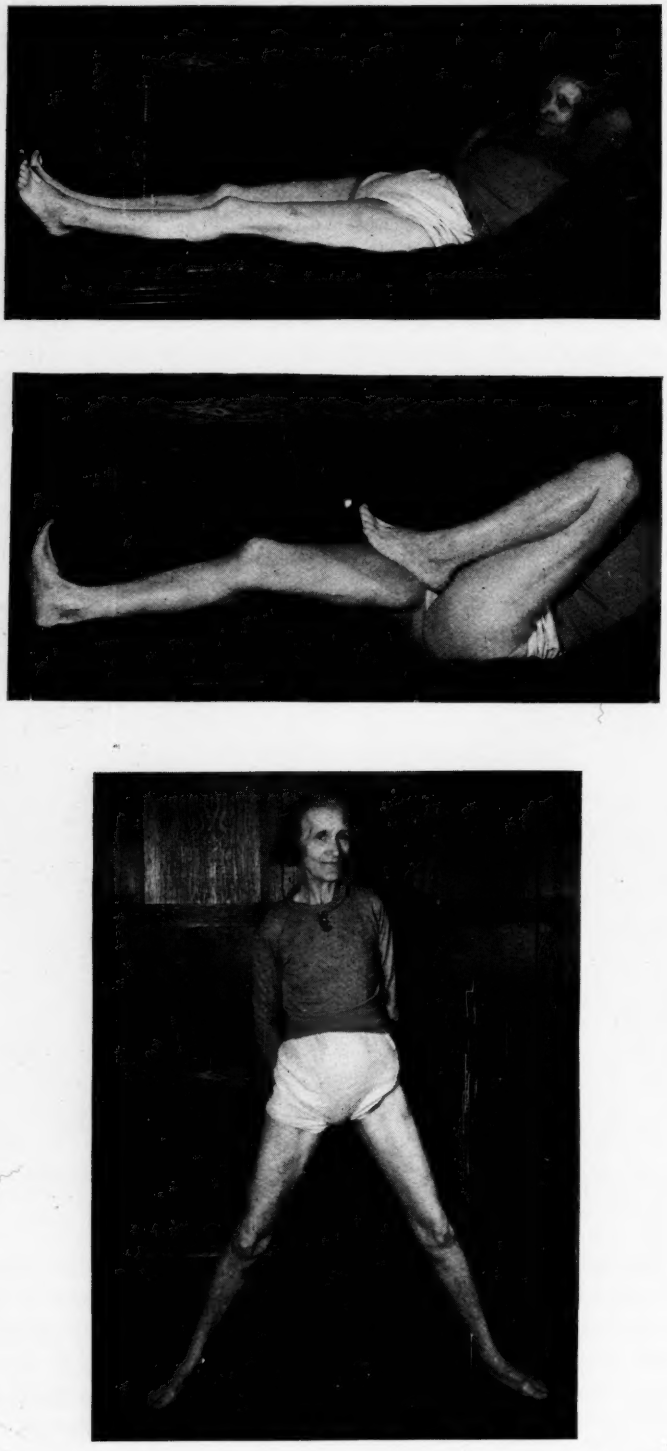

FIG. 582.—An almost normal range of hip and knee movement may be anticipated if the fracture is perfectly reduced, and knee exercises are begun the day after operation.

Joint Stiffness.—Most patients have no difficulty in retaining a full range of flexion of the knee provided that active movements are begun immediately after operation. No patient in this series had less than right-angled flexion, and most of them recovered full movement (*Fig.* 582).

The range of hip movement depends on the accuracy of reduction. In one case there was serious external rotation deformity and only 60° of hip flexion. The average range of hip flexion is from 90° to 160°, but it is always found, in cases free from arthritic change, that limitation of hip movement is due to inaccurate reduction. This provides one of the strongest arguments for the open operative method where the fragments are fully visualized, particularly in younger patients, and especially in working men.

Arthritis of the Hip-joint.—Not all of the cases in the series have been under observation for a sufficient number of years to know whether osteo-arthritis will develop or not. In one patient operated on within a few days of fracture there is severe arthritis with a painful stiff hip-joint three years after operation (*Fig.* 583). A second patient, operated on six months after fracture, has an equally severe arthritis. A third, operated on within three weeks, has slight arthritic change with some pain. In all of these cases the fracture was subcapital and there had been radiographic evidence of impairment of circulation of the proximal fragment. The complication is not entirely within the control of the surgeon, but its incidence may be minimized by operating early, by avoiding unnecessary hammering of the joint, by avoiding early weight-bearing, and by removing the nail after consolidation of the fracture.

FIG. 583.—Subcapital fracture of the neck of the femur three years after nailing, showing degenerative arthritis due to necrosis of the head following avascularity. The incidence of this complication may be minimized by operating early, by avoiding undue impaction, and by prohibiting early weight-bearing.

RESULTS OF TREATMENT

The 64 cases which have been operated on are considered in two series, the first from 1930 to 1933, when the Smith-Petersen technique was mainly used (29 cases), and the second from 1933 to 1935, when the author's technique was followed (35 cases) (*Tables I* and *II*). The first series suffered from the disadvantages of inexperience, and moreover it includes failures due to the use of plated nails. On the other hand, with extended experience an increasingly high proportion of cases have been operated on, and less and less promising material has been accepted as suitable. Whereas in the first series operation was performed in only half of the total fractures seen, in the second series 85 per cent of the fractures were treated

by operation. These patients are not only old, the average age being 60, but they include many with serious constitutional disorders—chronic bronchitis, emphysema, circulatory impairment, auricular fibrillation, pernicious anæmia, enlargement of the prostate, and uræmia. Even so, successful results were secured in 91 per cent of cases in the second series.

The operation mortality-rate of the whole group of 64 cases is 6 per cent, a mortality which compares very favourably with the general mortality of this fracture treated by all methods. In 30 personal cases treated in earlier years before the introduction of the nailing operation there was a mortality of 14 per cent, and the combined figures of several voluntary and municipal hospitals in Liverpool and Manchester showed a mortality of 21 per cent in 61 cases. There can be no doubt therefore that Smith-Petersen's discovery has not only revolutionized the prognosis of this injury from the point of view of union of the fracture, but it is also to be regarded as a life-saving measure.

Table I.—Analysis of Results

Smith-Petersen Technique (1930–3).—

Total cases seen ..	59 (average age 57)
Cases operated on ..	29 (49 per cent)
Failed	4 (3 re-fractures, 2 plated nails)
Died	2
Bony union	23 (79 per cent)

Author's Technique (1933–5).—

Total cases seen ..	41 (average age 60)
Cases operated on ..	35 (85 per cent)
Excluding 3 atypical cases	
Failed	1 (sepsis)
Died	2
Bony union	29 (91 per cent)

Mortality of combined series—4 in 64 (6 per cent)

Table II.—Detailed Results in Both Series of Operations

Patient	Age	Site	Result	Comments
Smith-Petersen Technique (1930–3).—				
E. E.	61	Cervical	Non-union	Plated nail. Absorption of bone
F. M.	21	Cervical	United	
A. S.	69	Cervical	Non-union	Plated nail. Absorption of bone
T. S.	34	Cervical	United	
P. K.	40	Cervical	United	Two nails used
F. R.	15	Cervical	United	
T. P.	40	Cervical	United	
E. S.	61	Cervical	United	
C. M.	60	Cervical	United	
de B.	70	Cervical	United	Re-fractured. Re-nailed (3rd week)
M. H.	78	Cervical	United	
M. M.	79	Basal	United	
J. S.	42	Cervical	United	
C. D.	56	Cervical	United	
E. W.	57	Cervical	Non-union	Re-fractured. Plaster spica (after few days)

Table II.—DETAILED RESULTS IN BOTH SERIES OF OPERATIONS, *continued*

PATIENT	AGE	SITE	RESULT	COMMENTS
Smith-Petersen Technique (1930–3), *continued*—				
O. J.	49	Cervical	United	
C. S.	46	Cervical	United	
A. D.	57	Cervical	United	Head completely avascular. Slow union (12 months). Osteo-arthritis (*see Fig.* 582)
I. M.	65	Cervical	United	
M. W.	68	Cervical	United	
A. H.	70	Cervical	Died	Died 3½ weeks. Pneumonia
M. C.	78	Cervical	United	
F. M.	65	Cervical	United	
M. H.	67	Cervical	United	Re-fracture (5th week). Re-nailed (*see Fig.* 575).
E. F.	46	Cervical	United	
B. M.	64	Basal	Fibrous union	Nail too high in shaft
M. G.	55	Cervical	United	
B. J.	79	Cervical	United	
M. H.	65	Cervical	Died	Died 10th week. Flap sloughed. (Enlarged prostate, uræmia, auricular fibrillation)
Author's Technique (1933–5).—				
S. M.	52	Cervical	United	
M. B.	75	Cervical	United	
C. C.	75	Cervical	Died	Died 3rd day. Very feeble constitution
M. A.	72	Cervical	United	Pernicious anæmia. Recently cured tuberculosis of lumbar spine (*see Fig.* 578)
E. W.	57	Cervical	Non-union	Sepsis. Low-grade infection—sinus. Nail removed. Plaster spica
F. M.	20	Cervical	United	
W. H.	44	Cervical	United	
E. G.	47	Cervical	United	
J. P.	73	Basal	United	Guide broke off in pelvis. Extracted 3 weeks later, abdominal route (*see Figs.* 576, 577)
E. McC.	57	Cervical	United	
W. G.	60	Cervical	United	
E. R.	58	Cervical	United	
S. W.	45	Cervical	United	
P. B.	91	Basal	United	Local anæsthetic. Subcutaneous insertion with X-ray control
A. D.	59	Cervical	United	
M. A.	74	Cervical	United	
E. P.	58	Cervical	Died	Died 1st day. Shock
P. L.	33	Cervical	United	
L. H.	60	Cervical	United	
C. N.	45	Cervical	United	
A. L.	65	Cervical	United	
M. D.	65	Basal	United	
A. S.	75	Basal	United	Shaft comminuted. Plaster spica after 10 days
M. O.	70	Cervical	United	Epileptic. Very frail. Local anæsthetic and subcutaneous insertion with X-ray control
G. T.	72	Cervical	United	
?	60	Cervical	United	Pernicious anæmia. Blood transfusion before operation
J. K.	78	Cervical	United	
S. A.	65	Cervical	United	
W. G.	60	Cervical	United	Severe poliomyelitis same limb
E. A.	72	Basal	United	
J. P.	46	Cervical	United	

Table II.—DETAILED RESULTS IN BOTH SERIES OF OPERATIONS, *continued.*

PATIENT	AGE	SITE	RESULT	COMMENTS
Atypical Cases.—				
E. T.	48	Cervical	Uniting	? Osteosclerosis. Previous spontaneous fracture humerus. Neck of femur extraordinarily dense. Still under treatment (partly united despite absorption upper half of fracture). (*See* p. 800)
S. C.	60	Cervical	United	Paget's disease. Neck of femur split. Nail removed. Plaster spica 6 months. United. (*See* p. 799)
M. C.	61	Cervical	United	7 months old. Aseptic necrosis of head. Nail gradually displaced. United after 18 months. Severe osteo-arthritis. Stiff painful hip. (*See* p. 789)

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TWO CASES OF CYSTADENOMA OF THE PANCREAS

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CYSTADENOMAS of the pancreas are sufficiently rare to justify the placing on record of individual cases. According to Miller (quoted by Huefer) only twenty-four cases were reported up to 1926, and there would appear to have been few additions to the literature since that time. The two cases that form the basis of this report came under the care of the writer within a comparatively short period and are the only ones that occur in the records of the Toronto General Hospital.

Case 1 (No. 081890).—Miss L. R., aged 70.

HISTORY.—Admitted on Oct. 15, 1933. Five years previously while massaging her abdomen to relieve constipation she noticed what seemed to be a small lump in the epigastrium. For three years the lump had remained barely palpable; it had enlarged slowly from that time. For the past two to three years she had been troubled with belching of gas and flatus, but during the past few months these symptoms had increased and had been accompanied by epigastric discomfort and water-brash. For one month she had had attacks of sharp pain that encircled the body at about the level of the umbilicus. An habitual constipation had increased recently. Two to three weeks previously her stools had been almost white for a few days. The appetite was poor, and eating was followed by discomfort and occasionally by vomiting. In the first eighteen months of the past two years there had been a loss in weight of about 6 lb., and in the past six months a loss of 40 lb.

ON EXAMINATION.—Although not in apparent distress, the patient was pale, very thin, and looked ill. A firm somewhat irregular mass filled the whole epigastrium. It was only slightly tender, moved with respiration, and did not seem to be attached to the liver, the lower edge of which was palpable 1 in. below the costal margin. Barium series and barium enema showed no intrinsic lesion of the stomach or colon. The pyloric end of the stomach and the duodenum were pushed downward and to the left, the displacement produced being such as would result from a retroperitoneal mass in the region of the head of the pancreas. The iodekon test showed a malfunctioning gall-bladder, and the leucocyte count was 10,000 per c.mm.

It was felt that the diagnosis lay between a cyst of the pancreas, a cyst of the liver, a carcinoma of the pancreas, and a mass of retroperitoneal glands, and that a laparotomy was necessary.

FIRST OPERATION (Oct. 19, 1933).—Spinal anæsthesia supplemented by gas and oxygen. Right paramedian incision. The mass was found to be a multiloculated cyst of the pancreas about the size of a large orange. It projected forward above the pylorus and first part of the duodenum and displaced the common

bile-duct to the right and the portal vein to the left (*Fig.* 584). The hepatic artery passed above and the pancreatico-duodenal artery round the right side of the tumour. The liver and gall-bladder appeared to be normal and the common bile-duct was not dilated. Removal of the cyst, although obviously difficult, seemed possible, and because the symptoms were increasing it was decided to attempt it.

The gastro-hepatic omentum was divided. The adjacent structures were separated slowly from the mass. The portal and superior mesenteric veins were especially difficult to free because they were bound to the cyst by innumerable bands of fibrous tissue which lay between the surface loculi and carried great numbers of small blood-vessels. A pedicle about 2 in. by ½ in. was finally obtained,

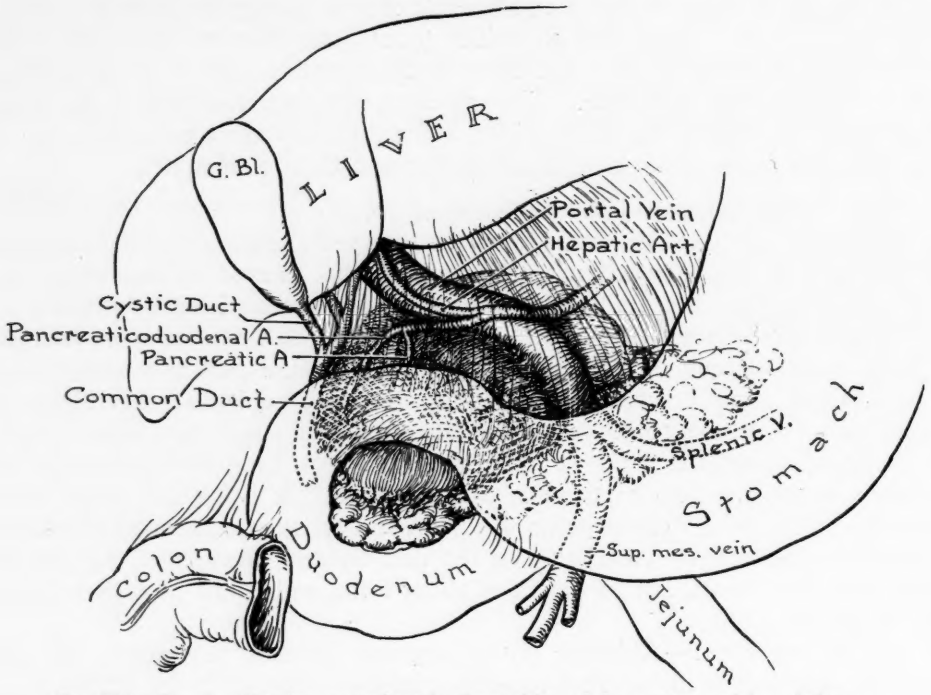

FIG. 584.—*Case* 1. Showing approximately the position of the tumour and its relation to surrounding structures.

but beyond this point the growth seemed to shade imperceptibly into pancreatic tissue. It was feared that further dissection might result in damage to the main duct of the pancreas. The pedicle was clamped, cut across, and oversewn with plain catgut. The abdomen was closed about a rubber drain containing plain gauze carried down to the bare area of the pancreas. Despite the fact that the operation had lasted for three hours the patient's condition was fairly satisfactory. A transfusion of 400 c.c. of blood was given.

There was remarkably little post-operative reaction, the maximum temperature being 101·5° and the pulse 100. The drain was removed on the fifth day. Considerable clear mucoid discharge containing pancreatic enzymes continued to come from the opening until Nov. 12, twenty-three days following operation. She was discharged on Nov. 20 free of symptoms but with a firm lump the size of a lemon in the region of the original mass.

RE-ADMISSION (Feb. 12, 1934).—The patient had remained free from symptoms until three weeks previously, when she had had an attack of generalized crampy abdominal pain. She was again well until three days before admission, when a similar attack of pain occurred, this time accompanied by vomiting. The temperature was 100°, pulse 100, and leucocyte count 23,000. The abdomen was slightly distended and in the upper right quadrant there was rigidity and tenderness. A tender mass could be felt in the right loin, but because of the rigidity and

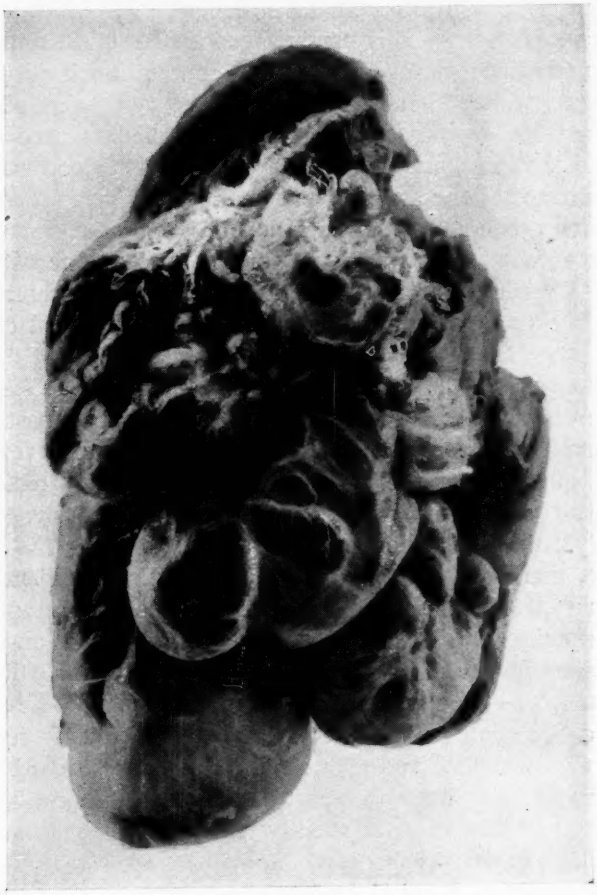

FIG. 585.—*Case* 1. Adenocystoma of pancreas. Photograph of gross specimen.

tenderness it could not be definitely outlined. A diagnosis of recurrent pancreatic cyst was made.

SECOND OPERATION (Feb. 15, 1934).—Gas and oxygen anæsthesia. A 2½-in. incision was made directly over the swelling. A pear-shaped mass, 3 × 3 × 5 in. was exposed, the base presenting. Opalescent fluid was obtained from it with a needle. The cyst was found to be unilocular and to have a wall about 3 mm. in thickness. It was marsupialized.

SUBSEQUENT PROGRESS.—Recovery was rapid and uneventful. The fluid discharged from the sinus was at first brownish in colour, but soon became clear

and watery in character; it contained pancreatic enzymes in small quantities. On March 11 the sinus was injected with lipiodol; no communication with the pancreatic ducts could be demonstrated. The patient was discharged on March 12 with a small drainage tube *in situ*.

Normal weight was regained rapidly. The escaping fluid gradually lessened in amount and became more mucoid in character. Temporary cessation of the discharge was on several occasions accompanied by pain. The opening finally closed about Jan. 15, 1935. There has been no further pain, no mass can be palpated, and the general health remains excellent.

PATHOLOGICAL REPORT.—This material consisted of a multiloculated cystic mass of tissue measuring overall $6 \times 6{\cdot}5 \times 7$ cm. to which was attached a small piece of normal pancreatic tissue (*Fig.* 585). The cysts were all thin-walled and varied from 1 to 3 cm. in diameter. They were filled for the most part with a clear watery fluid, but in some the fluid was slightly blood-stained. On cross-section the cut surface of the attached pancreatic tissue was reddish grey in colour and there were dense bands of pearly white fibrous tissue traversing it.

Microscopic sections (*Figs.* 586, 587) of the wall of one of the smaller cysts showed that it was made up of a rather loose areolar type of connective tissue lined on its inner surface by a very thin layer of cuboidal epithelial cells.

Case 2 (No. 090502).—Mrs. M. D., aged 29 years.

HISTORY.—Admitted on June 13, 1934. During the past three years the patient had suffered about once a month from an attack of severe colicky pain in the right upper quadrant of the abdomen. The pain radiated to the back and both shoulders and was accompanied by nausea and vomiting. She thought she had been slightly jaundiced following each attack. During the last attack, three weeks previously, there had been chills and sweating. The pain bore no relation to any particular type of food and there was little food intolerance. She suffered from a bitter taste in her mouth, belched considerable gas, and said she felt as though food "stuck in her mouth". She had recently lost 9 lb. in weight.

ON EXAMINATION.—The patient was thin and pale. The hæmoglobin was 72 per cent and leucocyte count 7200. The abdomen was scaphoid. A mass which appeared to be rounded and about 1 in. in diameter could be seen to descend with each inspiration along the upper part of the lateral margin of the right rectus. It was not readily palpable and was slightly tender. A diagnosis of chronic cholecystitis and cholelithiasis was made.

OPERATION (June 16, 1934).—Spinal anæsthesia. The abdomen was opened through a right paramedian incision. A mass the size of a large walnut presented within the curve of the duodenum. It was dark green in colour, firm but resilient as though cystic in character. It was found to originate from and to have largely replaced the head of the pancreas. A very thin layer of overlying pancreatic tissue was incised and the cyst easily enucleated with but slight bleeding. The pancreatic ducts were not encountered. The dead space was obliterated, the opening in the pancreas closed, and the peritoneum sutured over this with fine plain catgut. The gall-bladder was slightly thickened and there were a few pericholecystic adhesions, but no calculi. The ducts were normal. The liver showed moderate cirrhosis. The gall-bladder was removed and the abdomen closed without drainage.

Convalescence was uneventful and she was discharged on July 3, 1934.

FIG. 586.—*Case* 1. Microscopic section of tumour. (× 40.)

FIG. 587.—*Case* 1. Microscopic section of tumour. (× 80.)

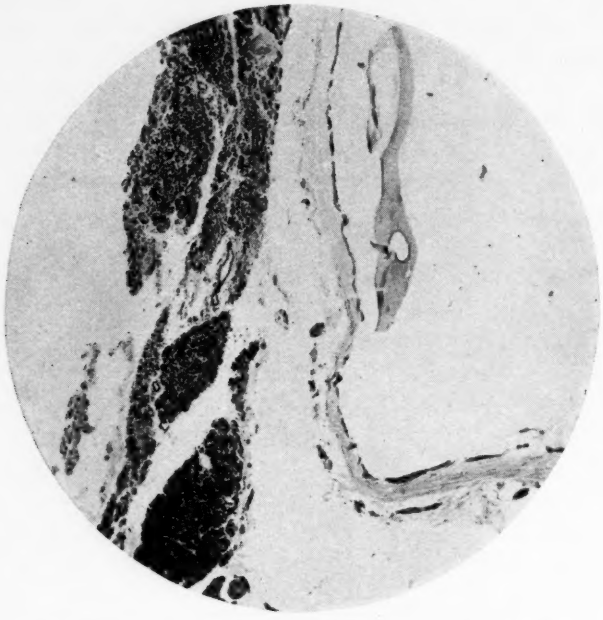

FIG. 588.—*Case* 2. Microscopic section of tumour. (× 40.)

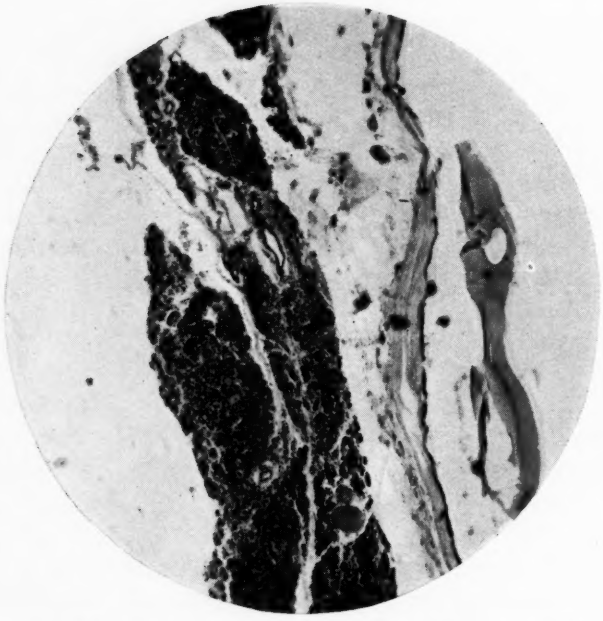

FIG. 589.—*Case* 2. Microscopic section of tumour. (× 80.)

PATHOLOGICAL REPORT.—The gross specimen consisted of a multiloculated cystic mass, roughly spherical in shape, measuring 2·5 cm. in diameter and weighing 6·8 grm. It was filled with a clear watery fluid.

Microscopically (*Figs.* 588, 589) the cyst wall itself was quite thin, being made up of dense fibrous tissue lined on its inner surface by a single layer of somewhat flattened epithelial cells. A portion of adjacent pancreatic tissue included in the section showed no pathological change.

For the pathological descriptions and the photomicrographs I am indebted to the Department of Pathology of the University of Toronto.

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PROSTATECTOMY WITH CLOSURE: ADDENDA AND SOME OBSERVATIONS

By S. HARRY HARRIS

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Two years ago the writer[1] described in this JOURNAL his operation of "Prostatectomy with Closure". Certain modifications have been introduced since that time as set forth below.

Gauze swabbing in order to obtain adequate visualization of the prostatic environs after prostatectomy has always appeared to me to be somewhat of an anomaly. Recently I have succeeded, with the assistance of the Genito-Urinary Manufacturing Company of London, and of Leiter of Vienna, in providing suction through the posterior blade of my electrically-lighted self-retaining retractor. In its latest form (*Fig.* 590, *a*, *b*) this instrument is of simple construction and only slightly heavier in weight than its plain-blade predecessor. It effects immediate evacuation of any overflow of blood from the prostatic loge.

In order to complete the visualization, a separate and most efficient metal tube-sucker has been constructed for me (*Fig.* 591). It is multi-curved in such a way that it may be insinuated by the assistant from any position into the prostatic loge with a minimum of inconvenience to the operator, so that, with the instruments I employ, vision at practically all times remains unobstructed. The necessity for gauze-swabbing has thus been eliminated and the operation time considerably curtailed.

The curved-tube sucker may be used with advantage even without the sucker-blade, but the combination of the two lends a nicety of technique not otherwise obtainable.

This dual suction at operation was first successfully employed by me at Glasgow and Edinburgh in October and in Vienna in November, 1935. Earlier in the year opportunities had been afforded me, in various London hospitals, of demonstrating my method of prostatectomy with immediate closure. Throughout my entire tour it was my experience that the majority of surgeons were content to submit patients to prostatectomy in whom the bladder had not been cleaned up to the extent to which we have been accustomed in the Urological Department of the Lewisham Hospital, Sydney, and which is readily attainable by the methods we have for several years practised and previously described in detail, both in this JOURNAL and elsewhere. Particular reference may, perhaps, be made here to our simple permanganate-of-potash–nitrate-of-silver method of bladder preparation, with the retained catheter connected by rubber tubing to a partially filled bottle at the bedside instead of into a urinal between the patient's thighs. It was possible on several occasions to demonstrate the efficiency of this treatment on patients who had had already a long period of preparation. The rapid conversion of a turbid urine into one with a crystalline appearance was very striking, enabling, as it did, the safe undertaking both of the plastic procedure on the bladder base and of the primary closure.

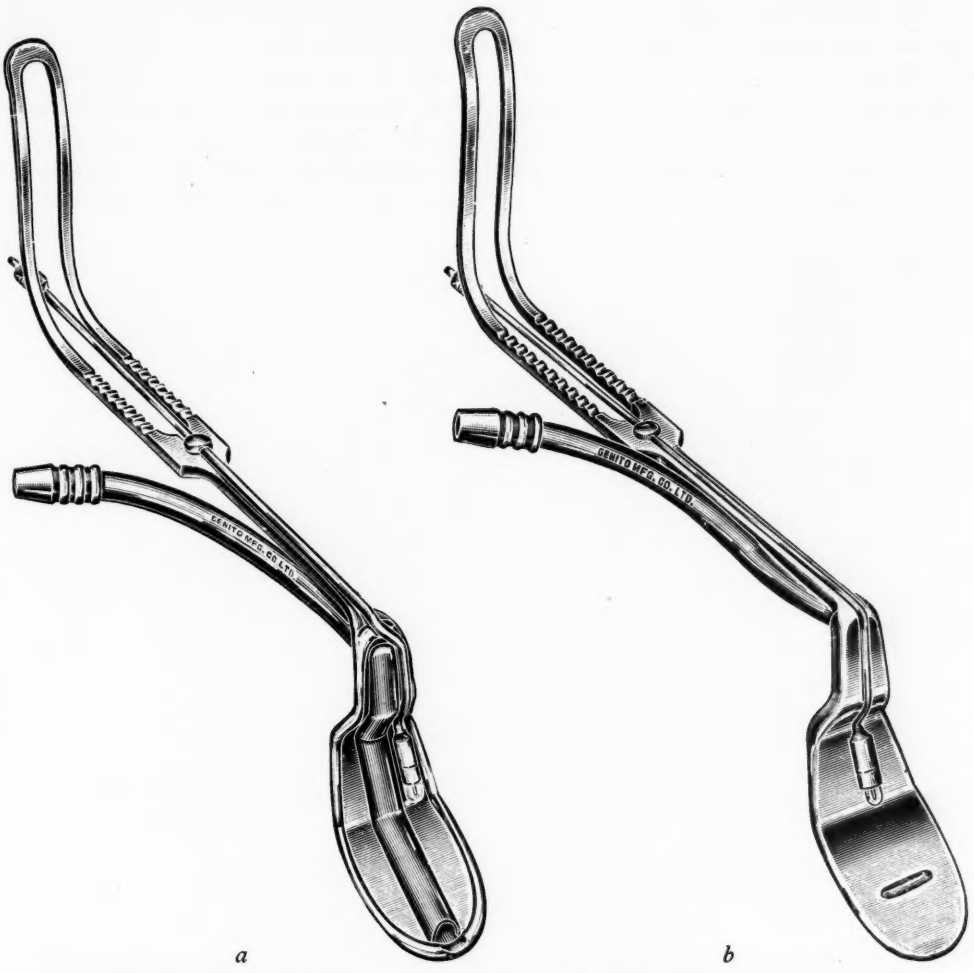

FIG. 590.—Posterior sucker-retractors to fit author's self-retaining frame, made (*a*) by Leiter, Vienna, (*b*) by the Genito-Urinary Manufacturing Company, London. Either may be employed with satisfaction. (*Reduced about ½ and drawn obliquely.*)

FIG. 591.—Individual sucker-tube made for the author by Leiter, Vienna. (*Reduced by more than ½.*)

So far as the *retrigonization* was concerned, this would appear to have been carried out by many surgeons in accordance with my original description. The simplified technique, however, described by me in the opening address on 'Prostatectomy' before the Royal Australasian College of Surgeons in Adelaide, in January, 1934, was, not unnaturally, unknown. With this technique it is *not* necessary to pick up with forceps the floor of the prostatic capsule before passing the boomerang needle in order to ensure a satisfactory retrigonization. The needle is now entered

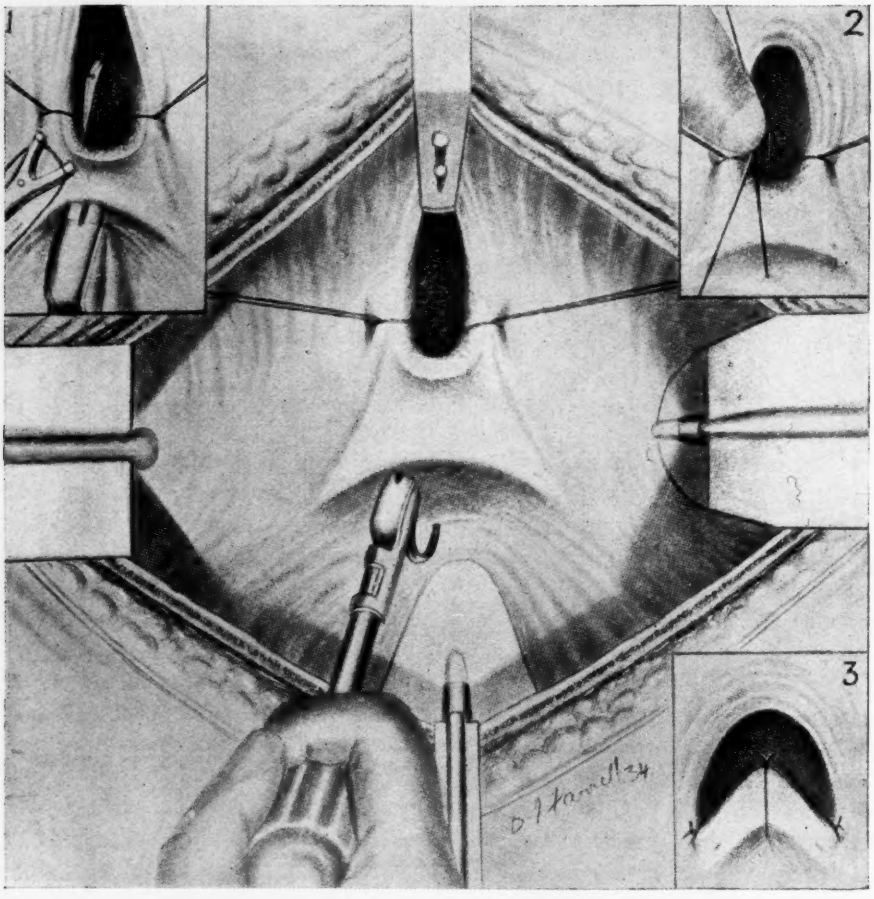

FIG. 592.—Illustrating the retrigonization of the prostatic urethra. The main figure shows the point of entry of the needle in the bladder base well down behind the inter-ureteric bar. *Insert* 1 shows the point of emergence of the needle well forward in the prostatic cavity and the suture carrier approaching the needle. *Insert* 2 shows the retrigonization suture being tied, with the forefinger of the left hand pressing the loop well down into the prostatic cavity. *Insert* 3 shows the retrigonization suture tied, sewing the trigone well down into the prostatic cavity, both obliterating the ledge in this situation and re-forming, at least in part, the new prostatic urethra. Two hæmostatic sutures only are illustrated in all figures for the sake of simplicity.

in the mid-line at the deepest part of the 'bas fond' (*Figs* 592, 593). It is, of course, necessary to visualize the ureteral orifices at this stage. No fear need then be entertained of compressing them when the suture is tied. The point of emergence of the needle is well down through the floor of the prostatic cavity (*Figs.* 592, 593). A wide and deep extent of tissue is, with ease and certainty, included in the bite of the needle. No attempt is, or ever has been, made by me to pick up in this bite the torn end of the prostatic urethra.

With regard to the *anterior transverse sutures*, there was a considerable degree of misconception. I consider the method and the site of insertion of these sutures as I have described them are vital features of the operation. With this technique there is no tendency to strangulation of the inverted prostatic rim with later sloughing and secondary hæmorrhage. The points of entry and of exit of these sutures are from 1 to 1½ in. from the free edge of the prostatic rim, the variation depending on the size of the prostatic cavity. The first, or more anterior suture, crosses the bladder base and the bottom of the prostatic cavity transversely and in a plane tangential to the anterior segment of the prostatic rim. This suture is immediately

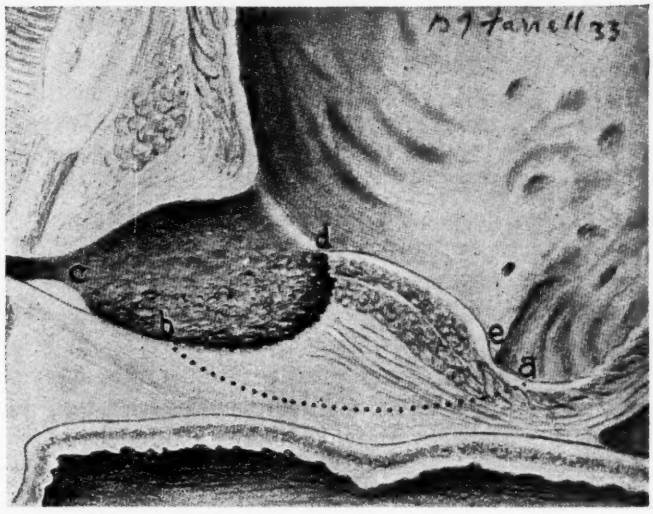

FIG. 593.—Illustrating the needle track, *a–b*, for the retrigonization suture. A sectional view of the prostatic environs after prostatectomy. *a*, Point of entry of the needle; *b*, Point of exit; *c*, Verumontanum; *d*, Torn apex of trigone; *e*, Base of trigone.

tied and converts the prostatic rim into a narrow oval. The second suture bisects this oval.

The catheter should on no account be passed before insertion of these sutures, as the catheter in the urethra presents an effective bar to the deep passage of the boomerang needle essential to secure for these sutures the necessary extent of bite. The tying of these sutures automatically brings about a broad, deep and smooth inversion of the prostatic rim and adjoining bladder base, while at the same time raising its level, thus securing: (1) Efficient obliteration of the prostatic cavity and venous oozing therefrom; (2) The re-formation in large degree of the side walls of the newly-formed urethra; and (3) A position for the new vesical outlet well below the level of the rest of the bladder base. The importance of the last-named both for the immediate catheter drainage and later for natural micturition cannot be over-estimated.

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EXPERIMENTAL SURGERY

INTESTINAL STRANGULATION*

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THE problem of 'acute intestinal obstruction' has for many years stimulated clinical and experimental investigation, yet it is a depressing fact that the mortality-rate still fails to show any great decrease commensurate with the effort which has been expended on this problem. The series of 1000 cases collected from the literature over the ten-year period 1888–98 by Charles L. Gibson[1] showed an average mortality of 43·2 per cent. Recently Christopher and Jennings[2] have published a composite series of 2345 cases, including the above, with an average mortality of 46·5 per cent. Recent series which exclude paralytic ileus and mesenteric thrombosis show a mortality in the region of 31 per cent—such as the series of Souttar[3], 3064 cases, mortality 32 per cent; McIver,[4] 335 cases, mortality 31 per cent. One great lesson may be learned from a study of the statistical tables —the extreme urgency of the earliest possible diagnosis and treatment in reducing the mortality. For the rest they provide a great stimulus to investigation. It cannot be too strongly insisted that the term 'acute intestinal obstruction' is only a general one, covering a variety of pathological conditions, in some of which at least, *the obstruction per se is the least important factor*. Insistence must be laid on the correlation of clinical, experimental, and statistical findings with a definite type of pathological lesion, and the application of the results obtained in the investigation of one group to that group only, and not to the subject as a whole.

It has long been recognized clinically that a difference exists between intestinal obstruction and acute intestinal strangulation. They differ in their pathology, symptomatology, and mortality-rates. In both conditions the gut is usually obstructed, but in the former group—intestinal obstruction (due to causes such as neoplasms, gall-stones, foreign bodies, enteroliths, kinks, and band compression) —obstruction occurs without circulatory involvement. Such cases, excluding neoplasms, represent about 20 per cent of the total (Hausler and Foster[5]). McIver[4] reports 37 per cent in a series of 395 cases. Patients with such a condition *after a period of days* may exhibit an almost normal temperature, pulse-rate, and blood-pressure. Their general condition is good, and, despite pain and distension, their ultimate collapse is delayed for a period which varies with the position of the obstruction, only occurring early if this is high in the region of jejunum or duodenum, and delayed possibly for weeks if it is in the large intestine.

In the second group—acute intestinal strangulation (due to such causes as

* The basis of a Hunterian Lecture delivered at the Royal College of Surgeons of England, on Feb. 10, 1936, by the former author.

volvulus, internal strangulation, hernia, intussusception, band strangulation, etc.)—the essential pathological feature is venous strangulation of the intestine. Such cases represent 80 per cent of the total in Hausler and Foster's series and 63 per cent in McIver's[4] series. Sir David Wilkie[6] has said: "We are all familiar with the striking picture of shock and collapse which follows hard on the onset of strangulation of the intestine." In a period of hours the blood-pressure and body temperature fall profoundly, the pulse is rapid, the skin cold and clammy, and, if left untreated, such a patient may well be dead in a period at which a patient with simple obstruction at the same level would still be in good condition. McIver[4] regards this factor of strangulation as being of greater importance than the position of the obstruction. "It is generally recognized that obstructions of the colon are less fulminating than those of the small intestine. Of course, however, if interference with the mesenteric circulation be present, death will occur as quickly in one situation as the other."

As regards the mortality-rate, McIver, in a series of 156 cases, gives the percentage of mortality as 53 where there is interference with the mesenteric circulation, and 37 in simple obstruction. Over a thirty-year series of 395 cases, the figures are 62 and 38 per cent respectively. One set of figures will be cited out of many which illustrate the urgency of early diagnosis—those of Christopher and Jennings[2] (*Table I*).

Table I

Duration of Symptoms before Operation	Mortality	
	Volvulus	Adhesions
	Per cent	Per cent
Less than 24 hours ..	0	0
24–48 hours	33·3	30
Over 48 hours	80	36
Not stated	100	14·4

From these facts it might be suggested that the greatest mortality arises where the gut is not only obstructed but also strangulated, and the process has been present for some length of time. Yet in the past by far the greatest volume of experimental effort has been directed to the investigation of intestinal obstruction without strangulation by such procedures as simple ligature of the intestine in continuity, double ligature at two points, division and re-suture without restoration of continuity, and formation of an isolated closed loop with restoration of continuity. In particular, attention has been focused on simple high obstruction of the small gut, the great infrequency of which may be judged from the fact that McIver could find only 3 examples without any vascular involvement in a series of 335 cases. It is therefore felt that any further investigation should be directed specifically to the problem of the circulatory collapse of intestinal strangulation, as opposed to simple obstruction. It is not intended to present a complete review of the exhaustive literature on simple intestinal obstruction, numerous résumés having appeared from time to time.[7, 8] Since in all our experiments the strangulation is associated with obstruction, it is important to realize what effects the obstruction alone might produce.

A few of the predominant points must therefore be summarized. Three theories have been advanced to account for the ultimate collapse of simple obstruction: (1) Reflex nervous action from the obstructed gut; (2) Bacterial invasion of the body from the same source; (3) A toxæmia.

Little real evidence has been advanced in support of the first theory, and it has been shown repeatedly that death may occur in the absence of any bacterial invasion of the peritoneum. In such cases cultures from the blood and viscera are sterile.[9, 10] The consensus of opinion is in favour of the third theory, but the origin and nature of the poisonous substance are by no means agreed upon. Clairmont and Ranzi[11] first showed that the content of an obstructed segment was toxic, but produced no evidence of its absorption. Cannon and Dragstedt[12] also hold that the toxin is elaborated from the content and is absorbed. Stone[7] holds a similar view. Whipple and his co-workers,[13] however, believe that the toxin results from an altered secretion of the intestinal mucosa, being partly secreted from it into the lumen, and in part absorbed into the blood-stream. As regards the nature of the toxin, there are also differences of opinion. Yet of the substances named, all might result from degradation of protein or similar large molecular substances. Sweet, Peet, and Hendrix[14] point out that such a process would result in the production of toxic proteoses and amines, while fat-splitting enzymes would produce from lecithin toxic cholines and neurines. We find that Whipple claims that the intoxication is due to a proteose.[15] Nesbitt[16] believes that neurines and cholines are significant, and Gerard[17] states that the substance is histamine (which Abel and Kubota[18] believe to be normally present in the digestive tract). Stone is of the opinion that a whole series of such split products are present in obstructed bowel, and possibly active in producing the lethal result. How such substances appear in the content, whether from bacterial activity[17, 19] or digestive action, is uncertain. Whipple[13] believes that neither bacterial nor pancreatic activity is significant, but that a proteose is secreted from the mucosa. Eisberg and numerous other workers, however, have demonstrated a greater toxicity of the loop content in the presence of digestive enzymes, and increase in the survival time if pancreatic secretions are excluded. Can this be of significance in relation to the greater toxicity of simple obstruction at the higher levels of the intestine? That such a difference in toxicity exists is shown by the survival times. Whipple, Stone, and Bernheim,[13] using washed and closed duodenal loops with restoration of continuity, gave an average survival time of forty-eight hours. Von Baracz[21] showed that closed loops of ileum and colon were very slightly toxic, dogs living for weeks or even months.

Of great importance is the time of appearance of the toxin. In all cases of experimental intestinal obstruction without strangulation, a definite interval elapses before much fluid collects in the loop and a still longer period before toxicity can be demonstrated in the gut content—Stone[22] gives these periods as 20 and 36 hours respectively. A summary of the available figures is given in *Table II*.

The presence of a toxin within the gut lumen has no significance whatever, if there is not positive evidence of its absorption. Although the toxicity of the loop content on intravenous injection has been amply demonstrated, Whipple[13] has shown that the injection of a double toxic dose of loop content into the lumen of a second obstructed loop does not accelerate the fatal outcome, and goes so far as to state that nothing produced inside the lumen of the intestine can be concerned

in the intoxication of intestinal obstruction, as such substances cannot be absorbed through intact mucosa. Cannon and Dragstedt[12] agree with this selective protective action, but believe that with *distension* and its resulting circulatory disturbance and tissue necrosis, absorption does take place. Brooks, Schumacher, and Wattenberg[27] also regard distension and necrosis as important factors in absorption, though not necessary for the actual production of toxin which is formed within the lumen. If distension is such an important factor, we have an alternative basis of explanation of the difference between high and low obstruction, for Murphy and Brooks[33] have demonstrated the more rapid secretion, and therefore more rapid distension, of high loops as compared with those that are prepared lower down in the intestine.

Table II

Reference	Time of Appearance of Toxin	Site	Level of Obstruction
Whipple[13] ..	24 hours	Mucosa	High closed loop
Stone[22]	36 hours	Lumen	High closed loop
Braye[23]	36 hours	Lumen	High closed loop
Dragstedt[25] ..	48 hours	Lumen	High obstruction
Van Beuren[24] ..	48 hours	Lumen	Simple jejunal ligature
Von Albeck[43] ..	24–48 hours	Lumen	Level not specified
Clairmont and Ranzi[11]	5 days	Lumen	Level not specified
Kukula[42]	3–10 days	Lumen	Level not specified

Saito[44] gives the period as 43 hours to 5 days according to the level, the toxin appearing earlier in the higher obstructions.

That a toxin does in fact appear in the blood, whatever its source, has been demonstrated. In 1909 Sauerbruch and Heyde[28] by a cross-circulation experiment were able to produce toxic symptoms in a normal animal as the result of intestinal obstruction in another. Sugito[29] has shown that the blood from the mesenteric veins draining the closed loop of intestine in a dog, if taken at the point of death, contains a toxic substance. This substance, *which is of the nature of a proteose*, is toxic on intravenous or intraperitoneal injection, in contrast to control injections of normal blood, and may be recovered from the spinal medulla, suggesting that it possesses a special affinity for nerve-cells. Scholefield[30] has confirmed the presence of this substance—again only in the terminal stages of closed-loop obstruction. It will be noted that in both these cases the experiments were conducted on closed loops, which are especially favourable to the occurrence of distension and its associated circulatory disturbances and necrosis. In Scholefield's post-mortem records such changes are specifically mentioned in 3 out of 4 cases. Although South and Hardt[31] believe that distension is not essential in the production of toxæmia, the recent work of Taylor, Wild, and Harrison[32] demands great attention. Introducing a rubber tube surrounded by a balloon into the intestinal lumen, they were able to produce distension of the gut without obstruction. X-rays demonstrated the passage of barium meal through the tube, but on distension of the balloon to a pressure of 100 mm. Hg, the animals succumbed in twenty-four hours with all the symptoms of experimental obstruction. These observers believe the distension produces this result through a nervous mechanism. A pressure of

100 mm. Hg, however, must necessarily cause venous occlusion, circulatory disturbances, anoxemia, and resulting changes in the tissues. In fact, it may produce certain of the characteristics of strangulation, which, as we shall show later, results in the rapid production of toxic substances and their appearance in the blood. It is, therefore, possible that the toxin which is found in the blood in the later stages of closed-loop obstruction may have had its origin in one of two sources: (1) Autolytic changes in the tissues of the gut wall itself—possibly resulting from anoxæmia, secondary to distension; or (2) The absorption of substances formed in the intestinal lumen.

Certain biochemical changes also support the presence of a toxin in the blood. Cooke, Rodenbaugh, and Whipple[34] have demonstrated a rise in the non-coagulable nitrogen of the blood in the presence of experimental intestinal obstruction, reaching three to ten times the normal figure at the height of the intoxication. A similar rise was produced by injection of toxic proteose, which Whipple[13] believed was secreted by the intestinal mucosa. They also demonstrated a rise in blood-urea and blood-creatinin, which they considered was due to protein or tissue destruction rather than impaired elimination.

The occurrence of dehydration and dechlorination is well known. Hartwell and Hoguet[10] noted the marked dehydration which occurred in obstructed animals after seven days' survival, and which they attributed to the associated vomiting. The dechlorination has been variously ascribed to loss by vomiting,[35] or, as suggested by Herrin and Meek,[36] to increased secretion, the result of distension, with chloride loss into the bowel lumen. Haden and Orr[37] suggested that the chloride is in part used up in the neutralization of toxic substances in the blood. Much has been written of the importance of dechlorination and dehydration in determining the fatal outcome, but experiments in which a fatal result has been directly produced by artificial dechlorination and dehydration are hard to find. Taylor and his co-workers produced this dechlorination by means of apomorphine vomiting, and conclude that their experiments in which "the blood chlorides were maintained at low levels for many days without serious effects resulting, must throw great doubt upon the primary importance of chloride loss as the cause of death in obstruction."

From these results it will be seen that the main evidence is in favour of death from simple obstruction being due to a toxæmia, the result of protein degradation occurring either in the bowel lumen or in the wall of the obstructed segment. A close corollary exists between the experimental and clinical picture in regard to the latent period before the onset of the toxæmia, forty-eight hours to five days, and to some extent in the greater severity of the high obstructions of the small intestine, although, as McIver has demonstrated, this latter point is less striking in the human than in the experimental animal.

It is in relation to this latent period that a difference exists between simple obstruction and acute intestinal strangulation. Murphy and Vincent[38] first demonstrated this point. Working with cats they prepared 12- to 15-cm. intestinal loops, from which the venous return was obstructed either by individual ligature of the veins or compression of the mesentery by a rubber ligature. They were able to demonstrate by blood-pressure records that the height of the toxæmia was reached in a period of four to six hours, at which time animals with simple obstruction (i.e., without venous obstruction) were quite unaffected. If, however, the

arteries were occluded as well as the veins, producing total anæmia, there was no such rapid intoxication. These results were confirmed by survival experiments. The results are shown in *Table III*.

Table III

Lesion	Position	Survival Time
Simple obstruction ..	High	4 days
	Low	7 days—killed
Total anæmia ..	High	3 days
	Low	2 days
Strangulation ..	High—5 cases	23–26 hours
	Low—4 cases	10, 17, 26, and 48 hours

Such figures emphasize not only the great severity of strangulation as compared with simple obstruction, but also the fact that when the gut is strangulated there is no evidence that a lesion high in the small intestine is more rapidly fatal than one situated at a lower level. From the figures given above Murphy and Vincent concluded, therefore, that blockage of the venous return was the most important factor in the production of this rapid intoxication. They were able to show that venous occlusion produced marked destructive changes in the gut mucosa within a period of four to five hours, such changes being absent in total anæmia. In their view death was due to the absorption through the damaged mucosa of bacterial toxins from the gut lumen, which entered the circulation through the lymphatic system.

Much has been written of bacterial toxins in relation to acute intestinal obstruction, especially those of the *Bacillus welchii*. The work of McIver, White, and Lawson[39] at least disposes of the *B. welchii* group. Working with cats they prepared 20-cm. strangulated loops by individual ligation of the mesenteric veins. Such animals had a survival period of eighteen to thirty-two hours. The investigation showed that the *B. welchii* present in the loop were mostly of an avirulent strain, that cats immune to *B. welchii* toxin succumb rapidly to strangulation, and that there was no evidence that life could be prolonged by the administration of antitoxin. Death occurred before there had been more than a slight fall in the blood-chloride content. It appears that in intestinal strangulation we can attribute death neither to *B. welchii* toxæmia nor to hypochloræmia.

Foster and Hausler[40] attribute death to different causes according to the length of the gut involved. They produced strangulation of various lengths of intestine in dogs by a rubber ligature surrounding the mesentery, and found that the survival was inversely proportional to the length of loop affected.

5-in. segment—survival 29 hours
15-in. segment—survival 22 hours
26-in. segment—survival 8·5 hours, etc.

They attribute death in animals with long-loop strangulation to shock—the result of distension and violent peristalsis together with loss of fluid from the circulation into the strangulated segment. When a short loop was affected the initial shock

was insufficient to account for death, which resulted later from the absorption of toxins, believed by them to be of bacterial origin, from the peritoneal cavity. By enclosing the strangulated segment in a rubber bag they were able to prevent this absorption into the circulation and to prolong life. They again point out that hypochloræmia cannot be a lethal factor in strangulation, as death takes place in the presence of almost normal blood-chlorides. Holt,[41] in repeating this work, came to similar conclusions, and in particular laid emphasis on the fact that death in long-loop strangulation was due entirely to the fluid loss. In medium loops he agrees with the conclusions of Foster and Hausler given above, except that the toxin is produced by bacterial proteolytic action on the gut wall and is not derived directly from the content. Although his loops were prepared by individual ligature of mesenteric veins, the significance of which will be pointed out later, he regards the route of absorption of this toxin as being via the peritoneal cavity in the latter stages. In very short loops he attributes death either to peritonitis, the result of perforation aided by the exudation of toxic loop content, or, in the case of femoral hernia, which he regards as extraperitoneal, as due simply to the obstruction and its resulting dehydration. The toxic properties of the peritoneal fluid seen in the latter stages are attributed by him to the presence of a proteose.

EXPERIMENTAL METHODS AND RESULTS

Our investigation was taken up with the object of determining the relative importance of fluid loss and toxæmia in producing the circulatory collapse of intestinal strangulation in loops of all sizes, as it seemed to us improbable that a toxin, whatever its source, which eventually appeared in the peritoneal fluid, could be without significance before its arrival at this point, and might therefore contribute to the collapse even in long-loop strangulation. We have been led from this initial point to an investigation of the source, the absorption, and the excretion of the toxin, a study of its action, and the possible effects resulting from its absorption following the relief of a strangulated segment. The details of the various operative procedures will be given under their appropriate sections, but certain general considerations must be dealt with at this point.

Since, as already indicated, there seems little ground for believing that the toxicity of strangulated loops varies with the level of the lesion, we have made a practice of forming our short strangulated segments at approximately the middle of the small intestine. It became necessary in the course of the investigation to be able to form strangulations of greater or lesser severity. Two procedures were adopted to this end.

1. **Severe Strangulation : Non-viable Type.**—The length of small intestine to be strangulated was carefully measured, and obstructing ligatures were placed around the gut at the limits of the selected length, producing a closed loop. The marginal vein at the gut border was ligatured and the individual veins in the mesentery draining the affected segment were tied off, leaving the arteries patent. The gut in the affected segment was thus in a condition of extreme venous strangulation, but since the mesentery was not compressed, *the lymphatics leading from it were not occluded.* Such a segment gradually became cyanosed and the mesentery engorged. Within a period of twenty-four hours the gut lost its 'viability', although no actual gangrene was present. The lumen was in all cases distended

with dark hæmorrhagic fluid. Sometimes a few of the mesenteric veins were thrombosed. The peritoneal fluid was copious, markedly blood-stained, and possessed a characteristic odour. From the dark colour of the gut such loops are referred to in this paper as 'black loops' (*Fig.* 594).

2. Mild Strangulation: Viable Type.—A length of loop was again measured, and obstructing ligatures were tied lightly so as to compress the gut without cutting into the wall, threads being left long in order to anchor the rubber ligature. The gut and mesentery were then surrounded with a rubber band. The tension on this was adjusted until the veins appeared perceptibly full and there was slight congestion of the mesentery and bowel; the ends of the band were then stitched together. Such compression of course affected the lymphatics of the mesentery as well as the veins. The gut gradually became dark pink in colour, and retained its 'viability' at the end of twenty-four hours. In these cases, also, the lumen was markedly distended by fluid. Peritoneal fluid was copious, clear, and odourless. From the colour of the gut such loops are referred to as 'pink loops' (*Fig.* 595). In a few cases where the tension on the ligature had been too great, the character of the gut and peritoneal fluid at twenty-four hours approximated to the condition of the former non-viable group. In all survival cases the operations were conducted with full aseptic technique and ether anæsthesia was employed.

EXPERIMENTAL RESULTS

Survival Times

In our early results we were able to confirm the findings of Murphy and Vincent[38] on the contrast between simple obstruction, venous strangulation, and total anæmia. As controls single-ligature obstructions and simple closed-loop obstructions of the same length as the strangulated segments were used (*Table IV*).

Table IV

Lesion	Position	Survival Time	Remarks
		Hours	
Simple obstruction ..	Middle small intestine	240 approx.	Peritonitis at ligature, no vomiting
Closed loop 20 cm.	Middle small intestine	163	Post mortem—peritoneum clear. No distension of loop (vomiting marked)
Total anæmia 20 cm.	Middle small intestine	48	Gangrene, peritonitis. No fluid loss
Strangulation (black loop) 20 cm.	Middle small intestine	19	Peritoneal exudate as described, no infection

Comparison of the survival times of two series of strangulated segments of moderate length but different degrees of severity, prepared by the methods that have already been indicated, shows a marked difference of the survival period (*Table V*).

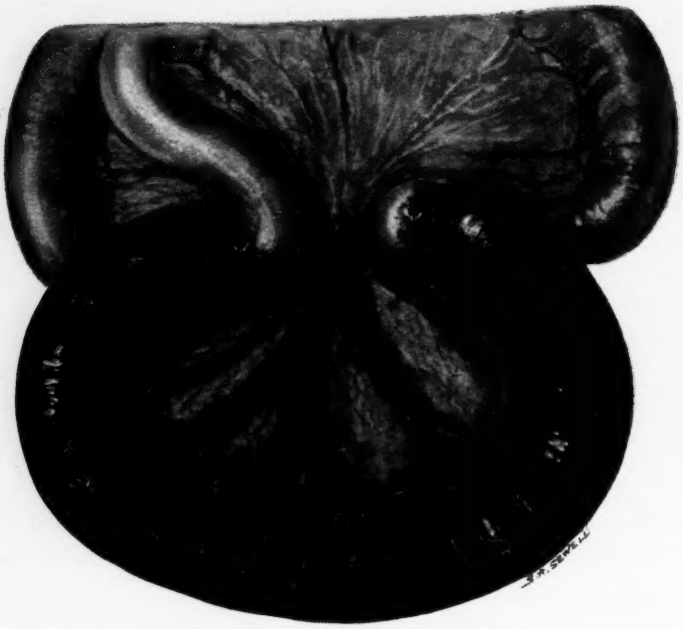

FIG. 594.—Non-viable strangulation of 24 hours' duration.

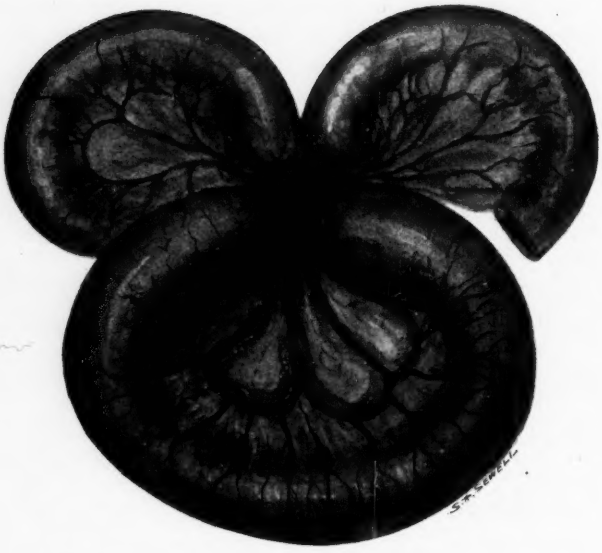

FIG. 595.—Viable strangulation, 24 hours' duration.

Table V

	Weight	Length of Loop	Survival Time
	Kg.	Cm.	Hours
Series I: **Black Loops.—**			
1	2·25	17	45
2	2·5	17	20·5
3	2·75	17	20
4	3·75	17	36
5	2·5	32	18
6	3·0	35	12
7	2·75	40	21
Series II: **Pink Loops.—**			
1	1·75	17	82
2	2·25	17	B.P. 110 at 22 hrs.
3	2·5	17	B.P. 70 at 22 hrs.
4	2·5	17	B.P. 100 at 48 hrs.
5	2·5	17	B.P. 100 at 48 hrs.
6	2·5	17	B.P. 110 at 48 hrs.
7	2·5	17	B.P. 60 at 48 hrs.
8	2·75	17	B.P. 70 at 22 hrs.
9	3·0	17	B.P. 110 at 48 hrs.
10	3·0	32	B.P. 90 at 20 hrs.

Since there is so marked a difference in the survival time where venous strangulation is present, it is well to consider its physiological consequences, for amongst them must be factors which accelerate the fatal outcome. They are obviously :—

1. Stagnation of blood in the local area.

2. Raised capillary pressure, resulting in increased tissue fluid formation and exudation into the bowel lumen and peritoneal cavity.

3. Distension of the gut.

4. Increased lymphatic fluid production with increased opportunities for absorption (provided the lymphatics are not compressed).

5. Stagnation anoxæmia and consequent changes in tissue metabolism progressing to devitalization of tissue.

The first two of these lead to loss of fluid from the circulation. The last two are of importance in relation to toxæmia.

The Fluid-loss Factor

Medium-length Loops.—As well as the survival time the fluid loss occurring was also noted in order to estimate the significance of this factor as a cause of death. The following method was adopted: The length of the segment to be strangulated was accurately measured at operation. At post-mortem the peritoneal cavity and intestines were swabbed dry with wool swabs of a known weight, which were then weighed. The strangulated segment was excised together with its mesentery, and weighed. From the figure obtained by the addition of these two weighings was subtracted the weight of the wool and the weight of a section of

normal intestine and mesentery from the same animal of a length equal to the original segment. The difference in weight is accounted for by peritoneal exudate, fluid in the gut wall and mesentery, and extravasated fluid in the lumen. The importance of measuring a length of intestine equal to that which was taken *before* strangulation is that a great increase in size results from the distension. Foster and Hausler put this increase as high as two to three times the original length. In our experiments the average increase was just under one-third of the original length. The weight obtained for the total fluid loss is expressed as a percentage of the body weight of the animal (*Table VI*).

Table VI

	Weight	Length of Loop	Survival Time	Fluid Loss / Body Weight	Per Cent Body Weight
	Grm.	Cm.	Hours		
Series III: **Black Loops.—**					
1	2250	17	45	$\frac{27}{2250}$	1·2
2	2250	17	21	$\frac{36}{2250}$	1·6
3	2750	17	20½	$\frac{43·5}{2750}$	1·7
4	2750	17	20	$\frac{34}{2750}$	1·2
5	3000	17	36	$\frac{22}{3000}$	0·7
6	3750	17	36	$\frac{50}{3750}$	1·33
7	2250	32	18	$\frac{31}{2250}$	1·5
8	3000	35	12	$\frac{78}{3000}$	2·6
9	2750	40	18	$\frac{102·5}{2750}$	3·7
Series III: **Pink Loops.—**					
10	2500	17	75	$\frac{22·5}{2500}$	0·9
11	1750	17	82	$\frac{35}{1750}$	2·0
12	2750	17	48	$\frac{12}{2950}$	0·44

These figures show conclusively that there is no constant relationship between the amount of fluid lost from the circulation and the survival period, nor does the difference in fluid loss account for the varying survival times seen in *Series I* and *II*. In *Series III*, No. 5, a black loop with fluid loss of 0·7 per cent body weight survives 36 hours. No. 10 in the same series shows a pink loop of equal length with fluid loss 0·9 per cent body weight surviving 75 hours. No. 11, a pink loop, with fluid loss 2 per cent survives 82 hours, whereas Nos. 1 to 4, black loops of equal length having a fluid loss from 1·2 to 1·7 per cent, survive from 20 to 45 hours. In certain cases those animals showing the larger fluid loss survive for a shorter period, though there are obvious exceptions to this. In no case is the volume of fluid loss sufficient to account for death. Blalock[45] has shown that it is necessary to remove amounts of blood representing at least 4 per cent of the body

weight in order to produce death from fluid loss alone. Removal of 2 to 3 per cent gives rise to tachycardia and slight stupor, and only a mild degree of shock. We have repeatedly confirmed this observation. Fluid loss, therefore, plays at most only an accessory rôle in the production of circulatory collapse resulting from medium loop strangulation.

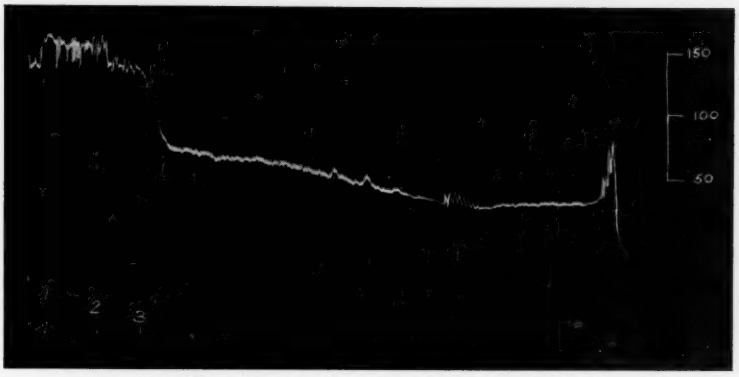

FIG. 596.—Carotid blood-pressure record showing effect of strangulation of entire small intestine of cat. Survival time: 2 hours. Fluid loss: 2·2 per cent of body weight. Sig. 1, Abdomen opened; Sig. 2, Ligature of veins; Sig. 3, Ligature of intestine.

Long-loop Strangulation.—Holt[41] has stated that in long-loop strangulation "there is no evidence of any toxæmic process, and death is due solely to the withholding of a large volume of blood from the circulation by the loop." To examine this point continuous blood-pressure records were made of cats anæsthetized with chloralose. The intestine was exposed and ligatures tied around the upper duodenum, and the ascending colon above the right colic vein. All collateral veins

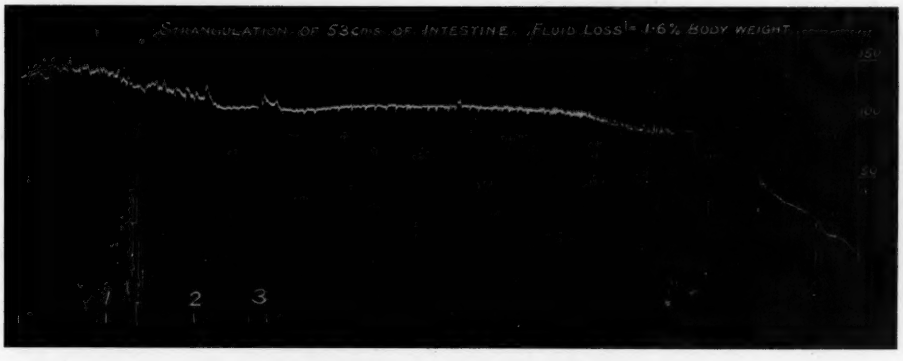

FIG. 597.—Carotid blood-pressure record showing the effect of strangulation of 53 cm. of small intestine. Survival time: 1 hour 10 minutes. Fluid loss: 1·6 per cent of body weight. Sig. 1, Ligature of mesenteric veins; Sig. 2, Ligature of collateral veins; Sig. 3, Ligature of intestine.

having been ligatured at these levels, the superior mesenteric vein was then tied below the junction with the splenic vein. A mass strangulation of the whole small intestine and portion of the upper colon was thus produced. This method will subsequently be referred to as 'superior mesenteric strangulation', and represents an extreme degree of long-loop strangulation.

Typical blood-pressure records obtained under these conditions are shown in *Figs.* 596, 597. At the end of the experiment the fluid loss was determined, by the following method. The peritoneal exudate was assessed in the manner stated above (p. 829), then the gut and mesentery were excised between the ligatures. The gut content, usually a few cubic centimetres of thick muco-hæmorrhagic fluid, was expressed and weighed. The gut and mesentery were then weighed. The total length of the intestine was then measured. After due allowance had been made for distension, the length obtained was multiplied by a weight factor derived from the examination of a series of 10-cm. lengths of normal small intestine. The weight so obtained was subtracted from the total weight of peritoneal fluid, gut content, strangulated wall and mesentery, and the figure for the fluid loss obtained (*Table VII*).

Table VII

	Weight	Length of Intestine	Survival Time	Fluid Loss	Per Cent of Body Weight
Series IV.—					
	Kg.	Cm.	Hours		
1	1·75	80	2	$\frac{39}{1750}$	2·2
2	1·5	53	—	$\frac{24}{1500}$	1·6
3	3·0	25	Killed at 7½ hours B.P. 30 mm.	$\frac{42}{3000}$	1·4
4	3·8	107	2¾	$\frac{64}{3500}$	1·8

It will be seen that here again the figure obtained for the fluid loss is in no case adequate to account for death even in the longest loops. The largest loss, that of 2·2 per cent, when produced by a rapid hæmorrhage, will cause an initial fall of pressure as shown in *Fig.* 596, which is the tracing taken from this animal, but hæmorrhage alone, as we have repeatedly found, is followed by a rapid recovery in the blood-pressure to a slightly lower level than the previous normal (*see Fig.* 600B), and is not succeeded by the gradual decline in pressure terminating in death as shown in this tracing. While admitting that the initial fall of pressure seen in certain cases can be due to fluid loss, we could not on this evidence admit that the fatal outcome was due to this factor alone. In view, however, of Holt's statement further experiments were conducted to settle this point.

Cross-circulation Experiments.—Two animals of approximately equal weight were selected. In one superior mesenteric strangulation was prepared by the method described above, a ligature being placed on the superior mesenteric vein but left untied. The carotid artery of the second animal was then connected through a Payr's cannula to the superior mesenteric artery supplying the segment to be strangulated, forming with it a surface lined completely with intima. The superior mesenteric vein was then tied and the clip removed from the carotid artery. The blood then flowed into the gut, which soon presented all the features of strangulation. Blood-pressure records were taken from the femoral artery of the supplying animal and the carotid artery of the recipient.

The blood-pressure of the 'supplying' animal shown in *Fig.* 598 remained steady despite the fact that it was suffering the entire fluid loss, which was subsequently estimated as 1·6 per cent of its body weight, a figure which corresponds well with those shown in *Table VII.* The 'strangulated' animal showed a progressive decline in blood-pressure comparable to that seen in *Fig.* 596, and died in one and a quarter hours, despite the absence of fluid loss. It will be noted that there is no rapid initial fall as shown in *Fig.* 596, which we have attributed to the

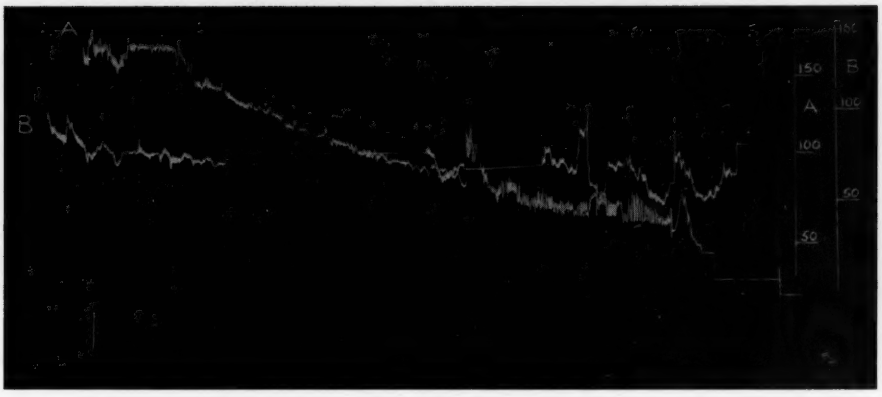

FIG. 598.—Cross-circulation experiment: Cat B supplying blood to strangulated intestine of Cat A. Death of strangulated Cat A in 1¼ hours despite absence of fluid loss.

loss of fluid. The significant feature is, however, the gradual decline in pressure which is maintained from the commencement of the strangulation.

Another similar experiment (*Fig.* 599) shows the supplying animal remaining at a steady pressure despite a fluid loss of 1·4 per cent of the body weight. The strangulated animal at first maintains its initial blood-pressure, later showing a progressive decline to death comparable to that seen in *Fig.* 597, despite the absence of fluid loss.

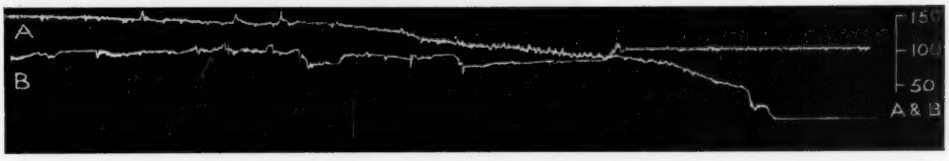

FIG. 599.—Cross-circulation experiment: Strangulated animal A dies after 2 hours. Fluid loss: 1·4 per cent of body weight of Cat B.

The experiment depicted in *Fig.* 600 is similar, but in addition illustrates the effect on blood-pressure of a large and rapid fluid loss, in this case amounting to 3·3 per cent of the body weight of the supplying animal. It will be seen that the initial fall of blood-pressure in the supplying animal is not followed by any progressive decline in pressure, the animal still showing a pressure of 90 mm. when the strangulated animal died.

These experiments prove conclusively that in long-loop strangulation death cannot be due to fluid loss alone. We therefore hold that whatever the length

of the loop affected, fluid loss plays no more than an accessory rôle in the production of the circulatory collapse of intestinal strangulation. The fatal result is in the main due to some other more important factor—either chemical or nervous.

In this paper we are concerned only with the chemical agency, consideration of a possible nervous mechanism being deferred.

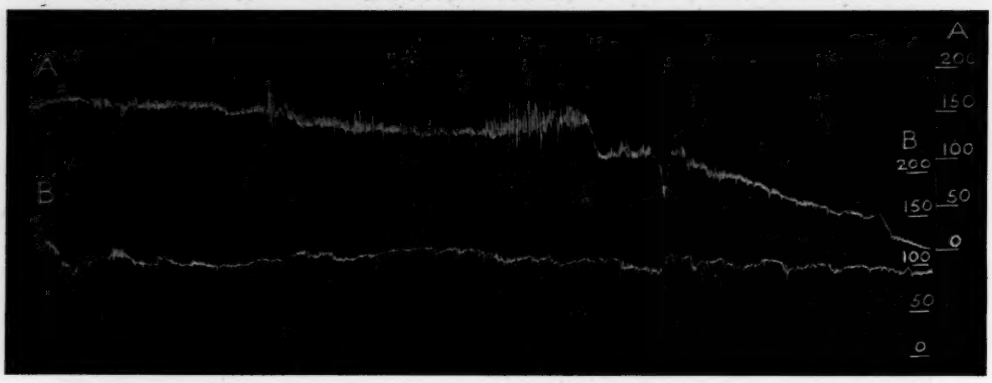

FIG. 600.—Cross-circulation experiment: Cat B (lower tracing) supplying blood to strangulated small intestine of Cat A (upper tracing). Death of Cat A after 2¾ hours. Fluid loss: 3·3 per cent of body weight of Cat B.

THE TOXIC FACTOR

Toxic Factor in the Peritoneal Fluid.—In order to confirm the toxicity of peritoneal fluid in the latter stages of strangulation of the black-loop type, and observe the result of its absorption from the peritoneal cavity, the following experiment was performed: A black loop of 17 cm. was formed and encased in a rubber bag. After 16½ hours the abdomen was opened and the 45 c.c. of fluid contained

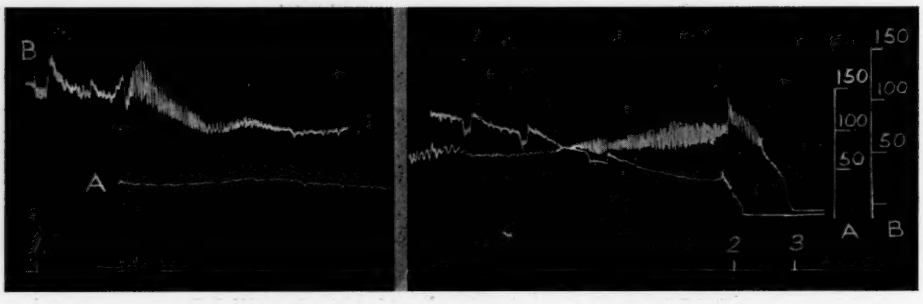

FIG. 601.—Effect of absorption of peritoneal fluid. Cat A (lower tracing), strangulated 16½ hours previously. Cat B (upper tracing), normal animal. Sig. 1, Peritoneal fluid and strangulated intestine of Cat A transferred to peritoneal cavity of Cat B; Sig. 2, Death of Cat B 3½ hours later; Sig. 3, Death of Cat A 20½ hours after original strangulation.

in the bag together with the loop were placed inside the peritoneal cavity of a normal animal. The abdominal walls of the two animals were sutured together and continuous blood records taken from each under chloralose anæsthesia (*Fig.* 601). The normal animal died in 3½ hours, having absorbed 30 c.c. of the exudate. The strangulated animal died shortly after, in a period of 20½ hours. This shows that the toxin appearing in the peritoneal fluid in cases of the non-viable type can

by itself without any fluid loss produce circulatory collapse and death in a normal animal.

Intravenous injections into the normal animal of peritoneal fluid derived from survival cases of the non-viable type, with one doubtful exception, always depressed

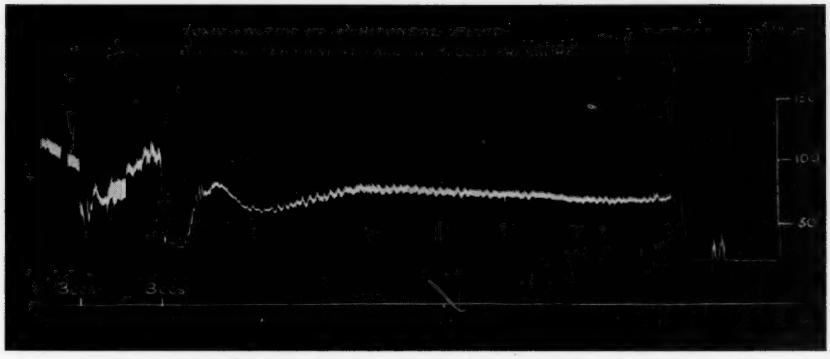

FIG. 602.—Peritoneal fluid. Depressor effect on blood-pressure of normal cat of intravenous injection of peritoneal fluid from a non-viable strangulation of 24 hours' duration. Sig. 1, 3 c.c. peritoneal fluid intravenously; Sig. 2, 3 c.c. peritoneal fluid intravenously.

the blood-pressure, provided the fluid was obtained a sufficient interval after the onset of strangulation. Following the initial fall in blood-pressure, the further character of the tracing varies. With small doses the blood-pressure usually returns to its original level. With larger doses the initial recovery does not take place,

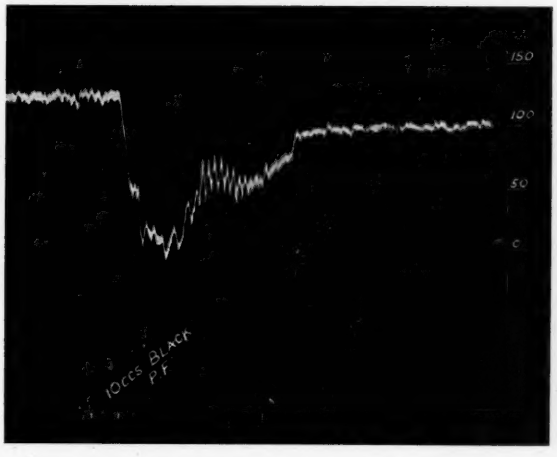

FIG. 603.—Peritoneal fluid. Depressor effect of intravenous injection into a normal cat of 10 c.c. peritoneal fluid from a non-viable strangulation of 23 hours' duration.

but instead the pressure returns slowly and remains stable at a permanently lower level. These reactions are illustrated in *Fig.* 602.

During the recovery phase the pulse was frequently observed to be slow and irregular. The presence of numerous dropped beats suggested that the toxic substance had some effect on the heart's action. (*Fig.* 603—*see also Fig.* 604.)

The permanent lowering of blood-pressure is of great significance in view of its relation to the clinical picture of intestinal strangulation.

Table VIII

Volume	Fresh or Post-mortem	Site of Injection	Effect	Duration of Strangulation
c.c.				Hours
Series V: **Viable Loops.—**				
60	Fresh	Intraperitoneal	Not toxic	22
20	Fresh	Intravenous	Not toxic	21
6	Fresh	Intravenous	Not toxic	48
6	Fresh	Intravenous	Pressor	48
2	Fresh	Intravenous	Pressor	29
10	Fresh	Intravenous	Pressor	23
2	Fresh	Intravenous	Pressor	24
Series V: **Non-viable Type.—**				
37	Fresh	Intraperitoneal	Death 3½ hours	16½
1	½ hr. P.M.	Intravenous	Depressor	12
1	12 hr. P.M.	Intravenous	Depressor	36
2	Fresh	Intravenous	Pressor ? ?	30
3	Fresh	Intravenous	Marked depressor	24
1	Fresh	Intravenous	Marked depressor	29
3	Fresh	Intravenous	Marked depressor	20
2	Fresh	Intravenous	Slightly depressor	20

A similar series of experiments using peritoneal fluid from 'viable' or 'pink' loops shows that there is no evidence of a toxic substance in such fluid. The intraperitoneal injection into a normal cat of 60 c.c. of peritoneal fluid collected in a rubber bag surrounding a 'viable' loop twenty-two hours after strangulation produced only a slight rise in blood-pressure. Intravenous injections of similar fluids (*see Figs.* 606, 619) are either without effect or give a mild pressor response. The difference in these results is shown in *Table VIII.*

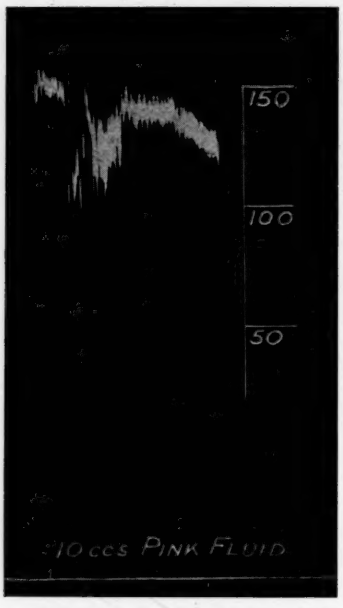

FIG. 604.—Peritoneal fluid. Slight depressor effect of intravenous injection into a normal cat of peritoneal fluid from a loop with doubtful viability of 24 hours' duration.

It is obvious, therefore, that a toxic substance appears in the peritoneal fluid within twelve hours of strangulation of the black-loop type, but that in the pink-loop type even after forty-eight hours of strangulation the peritoneal fluid has normal physiological effects, i.e., it is non-toxic.

We have investigated the time at which the depressor substance can first be recognized in peritoneal fluid. It can be demonstrated within sixty to seventy minutes of the production of a superior mesenteric strangulation. The fact that the peritoneal fluid in 'viable' loops does not contain a toxic substance within the time at which its presence can always be demonstrated in non-viable loops may explain the longer survival period already noted in

milder strangulations. A further point in the relationship between the toxicity of the exudate and the severity of the strangulation is brought out by the following observation: In two cases an attempt had been made to form viable loops, but,

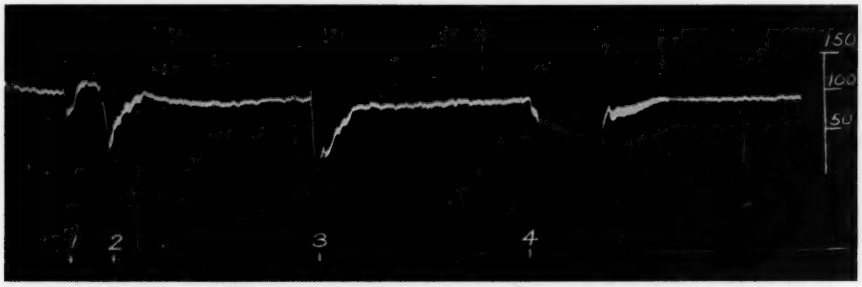

FIG. 605.—Effect of injection of peritoneal fluid and blood from the mesenteric veins of a non-viable strangulation. Sig. 1, Intravenous injection of 4 c.c. of peritoneal fluid; Sig. 2, Intravenous injection of 5 c.c. of peritoneal fluid; Sig. 3, Intravenous injection of 1 c.c. of mesenteric venous blood; Sig. 4, Intravenous injection of 9 c.c. of peritoneal fluid.

owing to excessive tension on the ligature, border-line cases of rather more severe strangulation and doubtful viability were produced at twenty-four hours.

The peritoneal fluid in these cases was slightly tinged with blood and gave a mild depressor reaction (*Fig.* 604). The severity of the strangulation, therefore, governs the time of appearance of a depressor substance in the peritoneal fluid.

Toxic Factor in the Gut Blood.—Viable and non-viable segments were constructed and after approximately twenty hours the abdomen was opened under anæsthesia and the blood in the mesenteric veins draining the loops aspirated and injected intravenously into normal animals. Injections of peritoneal fluid from the same cases were also made and the effects contrasted.

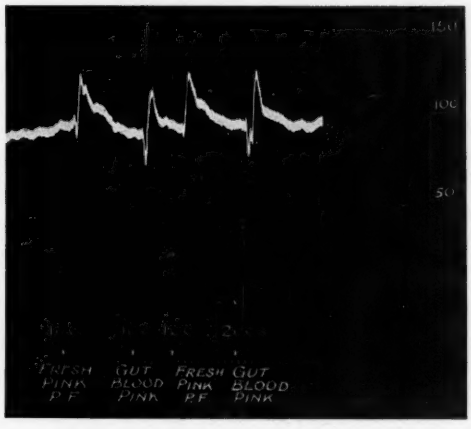

FIG. 606.—Effect of injection of peritoneal fluid and mesenteric venous blood from a viable strangulation of 17 cm. small intestine of 22 hours' duration. Sig. 1, Intravenous injection of 1 c.c. of peritoneal fluid. Sig. 2, Intravenous injection of 1 c.c. of mesenteric venous blood; Sig. 3, Intravenous injection of 1 c.c. of peritoneal fluid; Sig. 4, Intravenous injection of 2 c.c. of mesenteric venous blood.

Fig. 605 shows the results obtained by this method with material taken from a 17-cm. non-viable loop at twenty hours. A similar depressor substance is present both in the gut blood and in the peritoneal fluid. The injection of 1 c.c. of blood (Sig. 3) produces the same effect as the injection of 5 c.c. of peritoneal fluid (Sig. 2), suggesting a greater concentration in the former. The abnormal cardiac activity that has been already referred to is well shown during the recovery phase in this tracing (Sig. 4).

The injection of material derived from a viable loop at twenty-seven hours (*Fig.* 606) demonstrates that whereas the peritoneal fluid has a pressor reaction (Sigs. 1 and 3), as already described, the reaction of the gut blood in this case is

mildly depressor (Sigs. 2 and 4), though quantitatively less than in the case of non-viable gut (cf. *Fig.* 605, Sig. 3). A study of our results on this point shows that the gut blood always contains a markedly depressor substance in the case of non-viable loops, but that in viable loops the results are variable. The blood may contain either a smaller or equal amount of depressor substance in comparison with the blood of non-viable segments.

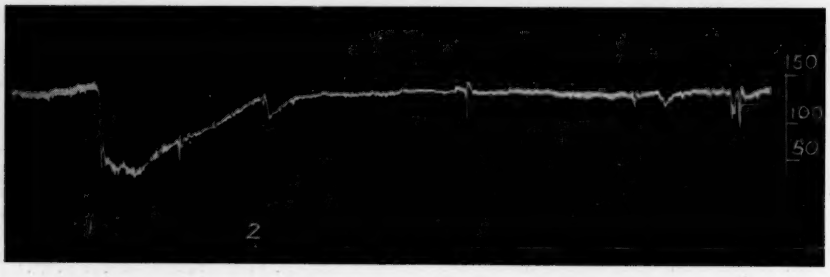

FIG. 607.—Effect of intravenous injection into normal cat of mesenteric venous blood from a very mild strangulation of 17 cm. of small intestine of 20 hours' duration. Sig. 1, Intravenous injection of 10 c.c. of mesenteric venous blood (anticoagulant—novhirudin); Sig. 2, Control injection of novhirudin solution.

Fig. 607 shows a case in which the blood from a very mild strangulation (' pink loop ') after twenty hours contained a large amount of depressor substance. In one case of our series the gut blood of a viable loop at twenty-four hours gave a marked and pure pressor reaction; the reason for this variability has not yet been determined.

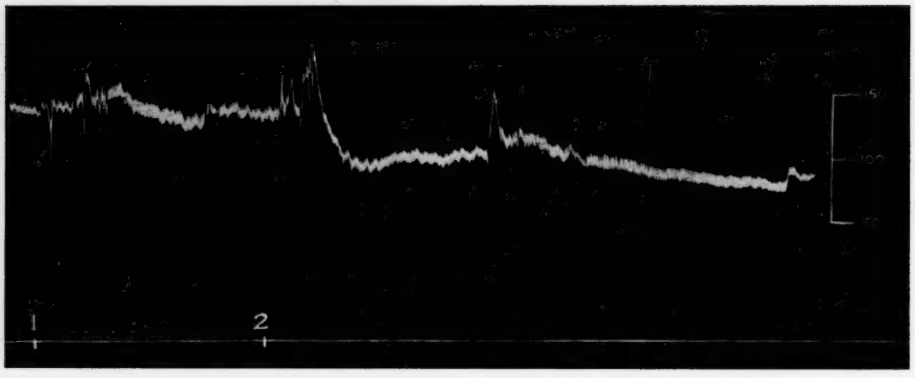

FIG. 608.—Blood collected from mesenteric veins after mass strangulation of small intestine for 1 hour and dialysed (10 c.c. dialysate equivalent to 1 c.c. of blood). Effect of injection of dialysate on blood-pressure of normal cat. Sig. 1, 10 c.c. of dialysate intravenously; Sig. 2, 50 c.c. of dialysate intravenously.

Time of Appearance.—In order to determine the period required before the appearance of depressor substance in the blood, superior mesenteric strangulations were performed and blood was collected by cannulation of the superior mesenteric vein and tested by intravenous injection in a normal animal. The presence of depressor substance was demonstrated in the venous blood in from thirty minutes to one hour from the onset of strangulation. Examination shows that this depressor substance is readily dialysable through a semi-permeable membrane (*Fig.* 608).

In this experiment blood was collected from the mesenteric vein into a weak anticoagulant solution (novhirudin) after one hour's strangulation and dialysed against ten times its volume of normal saline for twenty-four hours. It will be seen that 50 c.c. of dialysate, the equivalent of 5 c.c. of blood, are markedly depressor (Sig. 2), the effect being even more striking when we consider that the administration of 50 c.c. of saline by intravenous injection should by itself cause a very marked rise in pressure. This tracing also shows that whereas a small dose of fluid containing toxic material produces an effect with rapid recovery of the blood-pressure to normal (Sig. 1), a larger dose causes the permanent depressor effect already referred to (Sig. 2). In the examination of mesenteric blood from other experiments it was noted sometimes that the substance present appeared to have a profound effect on the heart of the injected animal. Small doses caused a slight fall in blood-pressure, but any attempt to give a large dose led to the immediate death of the injected animal by ventricular fibrillation. At post-mortem the liver was found to be engorged with dark blood. (*Figs.* 609 and 622, Sig. 5). We have met this phenomenon in three cases in our series.

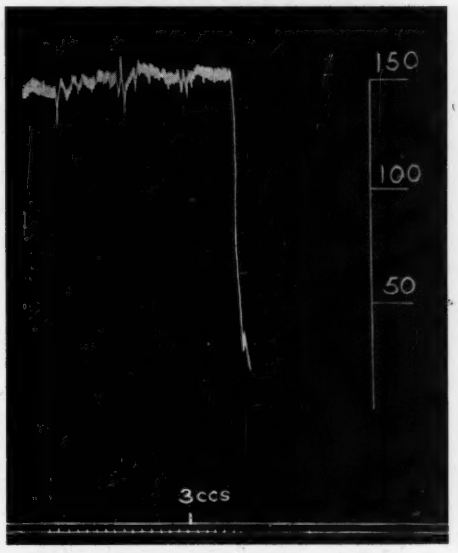

FIG. 609.—Injection of 3 c.c. of serum derived from venous blood from a strangulated segment of 20 cm. small intestine of 45 minutes' duration causes sudden death.

In view of the fact that Donomae and Feldberg,[46] in examining the portal blood of normal animals, had met with depressor effects in 3 out of 15 of their series, due to some substance other than acetylcholine, it was necessary to establish that the depressor substance observed in our cases had arisen as the direct result of strangulation. A superior mesenteric strangulation was prepared, the portal vein being exposed but left untied. A ligature was put in position above the point of junction of the splenic vein with the portal, and a cannula inserted into the splenic. Novhirudin was then administered to the animal and a sample of portal blood extracted from the splenic cannula. The portal vein was then tied and strangulation produced. After one hour's strangulation blood was again withdrawn from the portal vein. Intravenous injection of these samples into the normal animal showed that portal blood before strangulation gave a pressor reaction. The first sample removed after strangulation, which was merely the blood arrested at the upper end of the portal vein, was also pressor. The second sample which had been allowed to flow upwards from the gut was markedly depressor (*Fig.* 610). This result was constant in all five cases.

The character of the depressor effect seen in the tracings of different experiments showed variations, and in some cases alteration of the cardiac rhythm was noted. The question whether these differences are due to the presence of more than one substance or different concentrations of the same substance is under investigation. Our present results tend to support the latter view. It is definitely established, however, that a depressor substance appears in the venous blood within

thirty minutes to one hour from the onset of strangulation. The rapid appearance of the substance seems to rule out the possibility of a bacterial origin.

In order to determine whether the depressor substance is produced by the tissues of the gut wall by some abnormal process as the direct result of strangulation, or is merely a substance already existing in the lumen, which is absorbed under the new conditions, the following experiment was performed: The intestine was divided transversely immediately distal to the pylorus and above the ileocæcal valve. The pyloric and ileocæcal ends were ligatured and invaginated. Cannulæ were then tied in to the upper and lower ends of the isolated small intestine and connected to tubes through which the gut was irrigated. The lumen was washed through with 4 to 5 litres of warm water until the washings were quite clear. The water was then displaced with air and a variable quantity of warm normal saline introduced, usually 80 to 100 c.c. The rubber tubes were then clipped off, and by means of a water manometer connected through a side limb to the lowest cannula the pressure inside the lumen was recorded. After half an hour the saline was displaced and an equal volume of fresh saline introduced. The gut was then strangulated by ligature of the superior mesenteric vein and left for a further period of one hour, during which the pressure was noted. At the end of this time, the saline was again displaced from the lumen and a sample of blood removed from the mesenteric vein. Two samples of saline from the lumen were thus obtained, the first from normal resting intestine, the second from strangulated intestine. Each sample was filtered and the filtrate centrifuged. The first appeared clean, the second slightly blood-stained. These samples were then tested by intravenous injection in the normal animal. It was found in all cases that whereas the saline during the resting period was without any effect, the washing from the gut lumen was definitely depressor after one hour's strangulation. The samples of blood after one hour's strangulation were, with one exception, also depressor. These results may be seen from the *Table IX*.

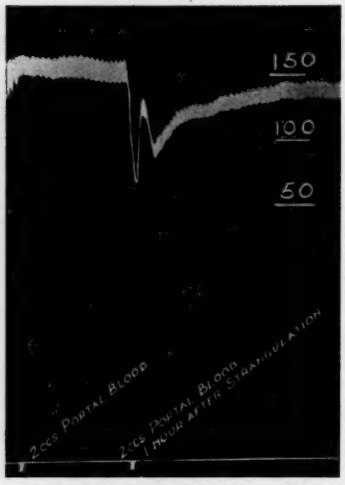

FIG. 610.—Effect of intravenous injection into normal animal of portal blood before and after strangulation. Sig. 1, Intravenous injection of 2 c.c. of portal blood before strangulation; Sig. 2, Intravenous injection of 2 c.c. of portal blood 1 hour after strangulation.

It will be noted that in no case does any depressor substance appear in the gut lumen during the normal resting period, nor could its presence be demonstrated in a control experiment in which simple obstruction was substituted for the strangulated period. *Fig.* 611 also illustrates these results, and in addition shows that the substance present in the gut lumen sometimes affects respiration, producing apnœa. The presence of a similar substance in the filtrate from the gut content of a viable loop excised at 24 hours is illustrated in *Fig.* 612 (Sig. 1), which also shows the marked depressor content of the venous blood in the same case (Sig. 2). These results appear to show that the depressor substance arises in the wall of the gut itself and passes from there both into the lumen and into the venous blood. Since it is not present in the lumen before strangulation, and since the rapidity of its formation in high concentration appears to rule out a bacterial origin, it would

seem that the toxin arises from the tissues of the gut wall as the result of intrinsic changes consequent upon venous strangulation. Such an origin for the depressor substance is well within the bounds of physiological possibilities. It is known that extracts from the wall of the intestinal tract contain depressor substances other

Table IX

	Irrigation	Resting Period, ½ Hour		Strangulated Period, 1 Hour		Gut Blood 1 Hour after Strangulation
		Pressure	Content	Pressure	Content	
1	4 litres till clear	4 cm. water	Pressor 2 c.c.	10 cm. water	Depressor 2·5 c.c.	Marked depressor 2·5 c.c.
2	3 litres till clear	2 cm. water	Pressor 2 c.c.	7 cm. water	Depressor 2 c.c.	Pressor 2 c.c.
3	4 litres till clear	1 cm. water	No effect 2 c.c.	6 cm. water	Marked depressor 2 c.c.	Depressor 2 c.c.
4	5 litres till clear	1 cm. water	No effect 2 c.c.	10–16 cm.	Depressor 2 c.c.	Marked depressor 4 c.c.

than histamine and acetylcholine. According to Euler and Gaddum[47] the muscle of the intestine is the source of depressor substance which gives blood-pressure reactions very similar to those already described in our experiments. Lim, Ling, and Liu[48] have prepared from intestinal mucosa an extract, the depressor effects

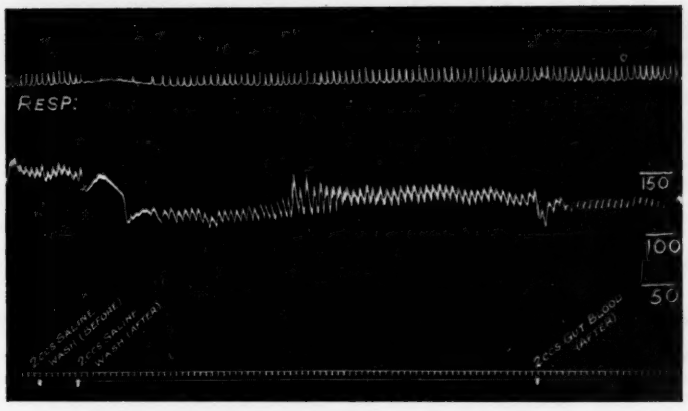

Fig. 611.—Effect of injection of intestinal contents before and after strangulation. Lumen of small intestine washed till clear. Filled with saline and left for half an hour. Saline removed and filtered; 2 c.c. of filtrate injected into test animal (Sig. 1). Intestine strangulated for 1 hour. Lumen of intestine washed with saline. Filtered. 2 c.c. of filtrate injected into test animal (Sig. 2); Sig. 3, 2 c.c. of portal blood 1 hour after strangulation.

of which are even more closely comparable to ours. This they believed to be a mixture of a small quantity of histamine with the Euler-Gaddum-Felix-Lange substance, and state definitely that its effects are not due to acetylcholine, adenyl compounds, kallikrein, or Major's non-guanidine depressor substance. We are not yet in a position to identify the substance or substances concerned in our reactions.

Distension.—It will be noted that in the period of strangulation in the above experiment the highest pressure produced varied between 14 and 16 cm. of water. This was purposely selected to accord with the pressures recorded in cases of intestinal obstruction in the human by Sperling and Wangensteen,[49] who demonstrated pressures of 14 to 18 cm. of *water* reaching a maximum of 30 cm. at the height of peristalsis. It is felt that the pressures of 80 to 100 mm. of *mercury* used by Taylor and his co-workers in the experiments already referred to are somewhat excessive, although McIver, White, and Lawson[39] have demonstrated pressures of 54 mm. of mercury in the strangulated loops of cats. That distension to a pressure of 80 to 100 mm. of mercury by itself produces significant results in the normal animal

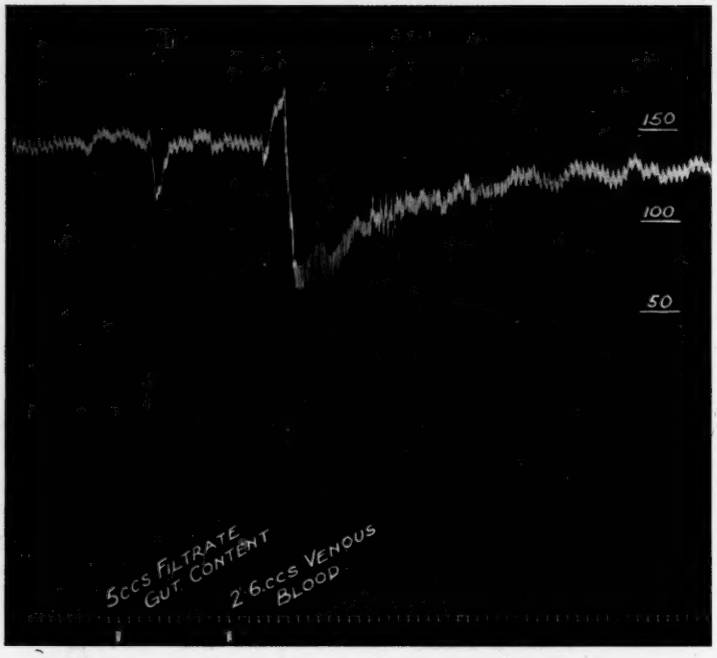

FIG. 612.—Effect of injection of filtrate of intestinal content and of venous blood from a 17-cm. viable loop of 24 hours' duration. Sig. 1, 5 c.c. filtrate of gut contents; Sig. 2, 2·6 c.c. mesenteric venous blood.

is undoubted. The precise mechanism by which it does so is at present under investigation. Our present results point to alteration in the degree of distension as the important factor rather than to simple distension at a constant pressure. This is of interest since peristalsis in the strangulated segment will obviously lead to rhythmic variations in pressure.

Distension in Normal Gut.—Fig. 613 shows the result of simple distension of a 17-cm. segment of normal gut to a pressure of 100 mm. of mercury. During the period that the pressure is maintained there is a rise in blood-pressure which is due to a nervous mechanism and can be abolished by section of the sympathetic and vagus nerves. Gradual relief of the distension is accompanied by a decline in blood-pressure to a lower level. On further distension and relief the same effects are produced. By this means the blood-pressure of a normal cat can be reduced to 90 mm. of mercury although death does not occur. Whether this is due entirely

to a nervous mechanism or the local production of depressor substance is under investigation.

Distension with Venous Obstruction.—In certain cases we have found that simple venous occlusion from a short segment of intestine without obstructing ligatures around the lumen may be survived for a period of several days or indefinitely,

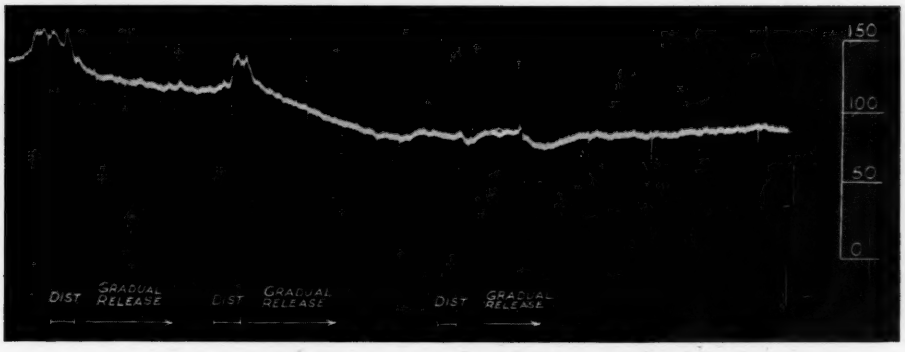

FIG. 613.—Effect on the blood-pressure of a normal animal of periodic distension of a segment of small intestine.

a partial venous collateral circulation being established. One animal had a blood-pressure of 120 mm. of mercury three days after such a procedure. On subsequent distension and relaxation of the affected segment a marked fall in blood-pressure and death resulted (*Fig.* 614). Rhythmic distension of the loop is, therefore, a factor of great importance. Whether its results are due to further production of the

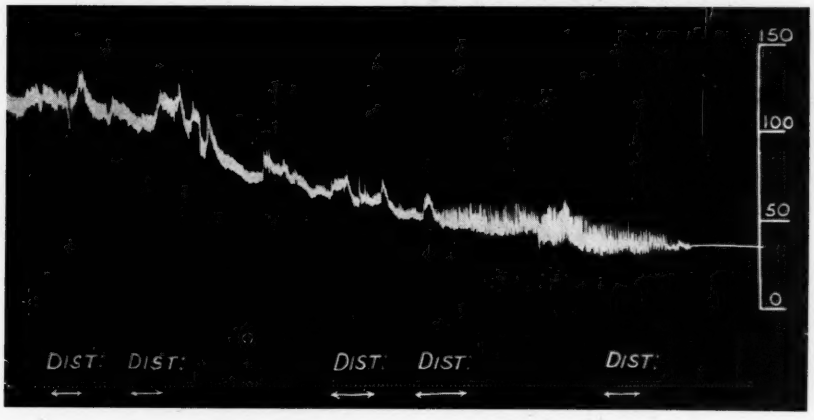

FIG. 614.—Effect on the blood-pressure of periodic distension of a segment of small intestine; following simple venous obstruction without occlusion of the lumen of the intestine performed 3 days previously.

depressor substance or to increased absorption from within the gut wall, or from the lumen, is at present under investigation. That increased lymphatic absorption of dye-stuffs results from distension of the intestine to 60 mm. of mercury pressure has already been demonstrated by Sperling and Wangensteen.[49] Absence of distension and diminished absorption may, therefore, allow the animal to survive

venous occlusion without obstruction, if the toxin is lost into the lumen. With a closed loop distension may promote absorption of depressor substance and hence allow it to produce its harmful effect. These conjectures, however, await confirmation. Distension is definitely an ancillary factor of great importance.

Absorption.—We have repeatedly observed at post-mortem that in every case where strangulation was produced by venous ligature alone, leaving the mesenteric lymphatics patent, as in superior mesenteric strangulation, and also in the formation of non-viable black loops, the large lymphatic channel in the mesenteric root and the thoracic duct have contained darkly-coloured or blood-stained fluid. A typical post-mortem appearance is illustrated in *Fig.* 615, which shows the condition of the thoracic duct in a superior mesenteric strangulation of two hours' duration.

FIG. 615.—Photograph of thorax of cat showing the thoracic duct containing dark-coloured fluid 2 hours after superior mesenteric strangulation.

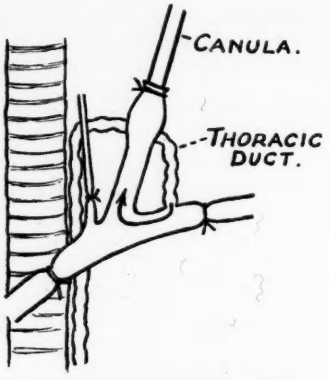

FIG. 616.—Method adopted in order to study the changes in the thoracic-duct outflow consequent upon strangulation.

To study the changes occurring in the outflow from the thoracic duct consequent on strangulation a fistula was formed (*Fig.* 616). The duct was identified in the left side of the neck and traced to its entry into the veins. The subclavian, innominate, and internal jugular veins were ligated on either side of the point of entry of the thoracic duct, causing the chyle to flow upwards into the external jugular vein. By insertion of a cannula into this vein, variations in the duct outflow were noted. On the production of a superior mesenteric strangulation a considerable increase in the rate of flow occurred, followed after an interval of up to thirty minutes by a marked change of colour from the normal pearly white to red (*Fig.* 617). This change of colour is due to pigmented serum and not to cellular elements. Simple coloration of the fluid does not, however, indicate a recognizable toxicity; depressor substance appears in the duct in recognizable quantity somewhat later, in our series between forty to seventy minutes of the onset of strangulation. The reason for this variation was not determined, but may possibly be a question of concentration. The experiment shows that in all those cases where the mesenteric lymphatics are patent,

absorption of depressor substance may occur directly into the thoracic duct, and thence into the systemic circulation. When, however, the lymphatics were occluded by compression of the mesentery, as in the formation of viable loops, we were not able to demonstrate depressor substance in the thoracic duct unless it were also present in the peritoneal fluid. In the latter case absorption appeared to be secondary from the peritoneum and not by a direct route from the gut lymphatics to the thoracic duct. This relationship is illustrated by the following results :—

Viable Loop	Gut Blood	Peritoneal Fluid	Thoracic Duct
18 hours	Depressor	Pressor	Negative
24 hours	Depressor	Pressor	Negative
Border-line			
24 hours	Depressor	Depressor	Depressor
23 hours	Depressor	Depressor	Depressor

In cases of the viable type, although the thoracic duct contains no depressor substance, the substance is present in the mesenteric lymphatics, being dammed

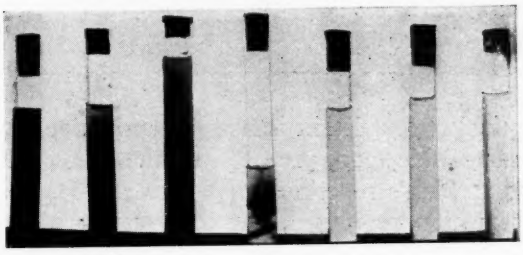

Fig. 617.—Photograph of fluid collected from thoracic-duct fistula showing the change occurring 15 minutes after the production of superior mesenteric strangulation.

back in them, and, on relief of the strangulation, appears in the thoracic duct (*see Fig.* 619).

In order to investigate the relationship of peritoneal absorption to the thoracic duct a few cubic centimetres of trypan blue were placed in the peritoneal cavity and seen to appear in the thoracic duct within twenty minutes. In view of the statement sometimes made that herniæ in which the neck of the sac is shut off should be regarded as extraperitoneal, a similar experiment was conducted. The tunica vaginalis was carefully dissected and firmly ligatured at the neck. Injection of trypan blue into the tunica led to the appearance of the dye in the thoracic duct within sixteen minutes.

Any toxic substance passing into the peritoneal cavity or hernial sac may, therefore, find its way into the general circulation via the thoracic duct. The possible routes of absorption in the various types of experiments conducted are summarized in *Table X.*

This table helps us to explain the longer survival period of the viable series already noted in *Series I* and *II.* Whereas at twenty-four hours the thoracic-duct fluid is non-toxic in viable-loop experiments, in the non-viable group it is profoundly depressor or toxic owing to continual mesenteric and peritoneal absorption. The importance of this factor may be judged from *Fig.* 618, which shows the effect of intravenous injection into the normal animal of 1·5 c.c. of thoracic-duct fluid obtained from a non-viable loop experiment in poor condition and with low blood-pressure

at twenty-three hours. The relationship between the severity of the strangulation and the survival time is obvious. The appearance of depressor substance in the peritoneal fluid, and hence its absorption through the thoracic duct, is governed by the severity of the strangulation. This is well illustrated in the viable and border-line cases, which, from their mode of formation by mesenteric compression, correspond

Table X

	Mesenteric Lymphatics	Peritoneal Fluid	Thoracic-Duct Fluid
Superior mesenteric strangulation	Patent, depressor	Depressor after 1 hour	Depressor 30–70 mins.
Black loops, non-viable—24 hours	Patent, depressor	Depressor	Marked depressor
Pink loops, viable—24 hours	Closed	Pressor	Normal
Border-line, i.e., of doubtful viability but produced by pink-loop method	Closed	Depressor	Depressor

most exactly to intestinal strangulation in the human patient. It will also be seen that where a black non-viable loop, prepared by the method already described, is enclosed in a bag, as in Holt's repetition of Foster and Hausler's experiment, not only is peritoneal absorption prevented, but compression of the mesentery by the neck of the bag occludes the mesenteric lymphatics. Such experiments, therefore, do not prove that absorption of toxin in these cases only occurs through the peritoneum, but that, provided toxic absorption is prevented by either the mesenteric or peritoneal route, life may be prolonged. We have repeated these experiments with variable results. In all cases where life was prolonged the thoracic-duct fluid was non-toxic. In the less successful cases the duct fluid contained toxic or depressor substances.

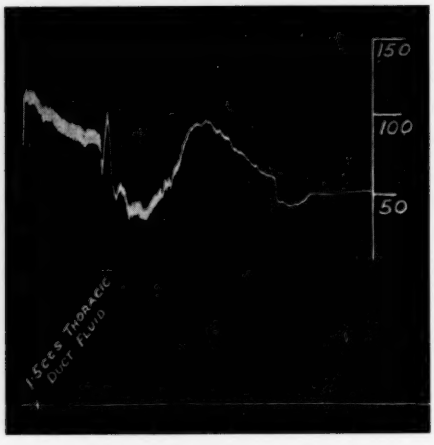

Fig. 618.—Thoracic-duct fluid. Effect of injection of 1·5 c.c. of thoracic-duct fluid collected from a cat with a non-viable strangulation of 23 hours' duration.

Relief of Strangulation.—Although the absorption of depressor substance before relief of the strangulation is undoubtedly important, the action of that fraction which remains dammed up in the veins and lymphatics of the strangulated loop is not without significance. On division of the mesenteric ligature in cases of the viable type depressor substance previously held up in strangulated veins and lymphatics is set free into the circulation. The lymphatics transmit the depressor substance into the thoracic duct as shown in *Fig.* 619, which illustrates the result of injection into the normal animal of material derived from a very mild 'viable'

strangulation at eighteen hours. The thoracic-duct fluid of the strangulated animal contained no depressor substance before the relief of strangulation (Sig. 2), since the peritoneal fluid was non-toxic (Sig. 1) and the lymphatics were compressed. Immediately after the strangulation was relieved depressor substance appeared in the thoracic-duct fluid (Sig. 3).

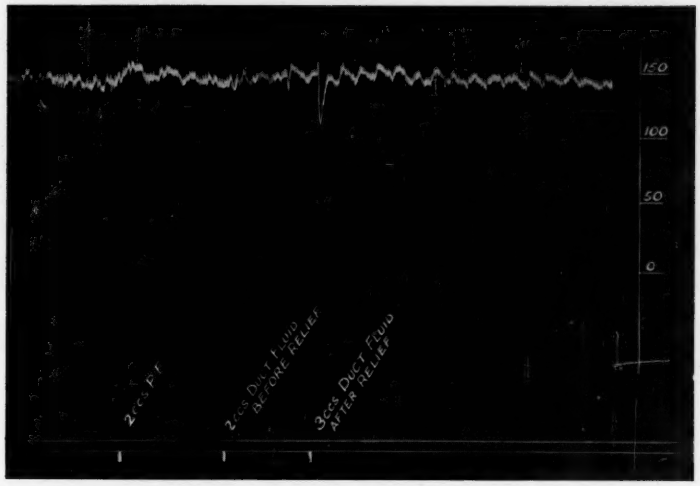

FIG. 619.—Thoracic-duct fluid. Relief of viable strangulation. Effect on the blood-pressure of a normal cat of intravenous injection of thoracic-duct fluid from another animal before and after relief of a viable strangulation. Sig. 1, Intravenous injection of 2 c.c. of peritoneal fluid; Sig. 2, Intravenous injection of 2 c.c. of thoracic-duct fluid before relief of strangulation; Sig. 3, Intravenous injection of 2 c.c. of thoracic-duct fluid after relief of strangulation.

When examining the fluid-loss factor we were repeatedly struck by the fact that relief of the venous obstruction, allowing the return of blood to the circulation, instead of improving the animal's condition always caused a further fall in blood-pressure. This, which would be inexplicable on the basis of the fluid-loss theory, is readily interpreted in relation to depressor substance in the veins. Such an experiment is illustrated in *Fig.* 620, where, following the relief of a very mild

FIG. 620.—Relief of strangulation. Decline of blood-pressure following relief of a viable strangulation of 17 cm. of small intestine of 26 hours' duration. Sig. 1, Relief of strangulation.

17-cm. viable strangulation at twenty-six hours, there is a progressive decline in blood-pressure from 120 to 60 mm. mercury in two hours.

Examination of the blood leaving a strangulated loop after relief of the strangulation shows that it continues to give a depressor reaction for thirty minutes or more, following relief of strangulation.

We believe that such re-absorption of depressor substance into the circulation accounts for the striking collapse shown in *Fig.* 621, where, following the relief of

a viable 17-cm. strangulation at twenty-two hours, the blood-pressure showed a rapid decline, and, despite the administration of saline and adrenalin, the animal died one and a quarter hours after the relief of strangulation.

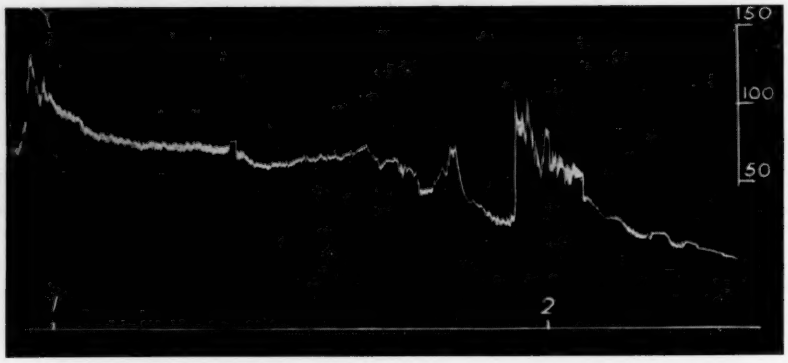

FIG. 621.—Relief of strangulation. Circulatory collapse following relief of viable strangulation of 17 cm. of small intestine of 22 hours' duration. Death of animal 1¼ hours later. Sig. 1, Relief of strangulation; Sig. 2, Intravenous adrenalin and saline.

Although evidence for the absorption of toxic material has been advanced, we have been unable to demonstrate the presence of depressor substance either in the carotid or heart blood of animals dying of experimental strangulation, nor could its presence be demonstrated in animals in which intravenous injections of toxic material had been performed. Whether this was due to rapid fixation or detoxication in the tissues, or rapid excretion from the body, is still the subject of investigation, together with the precise pharmacodynamic action of the substance on the heart and circulation. We have noted, however, that at post-mortem the liver frequently presents a nutmeg appearance, and a section shows evidence of fatty degeneration. Congestion of the renal glomeruli and fatty changes in the renal tubules have also been observed. Bearing in mind the demonstration of blood-pressure-reducing substances in the human urine by Adolf Bauer[50] and the work of Abelous and Bardier[51] on urohypotensine (*see below*), we were led to examine the urine of strangulated cats, in search of evidence of excretion of depressor substance.

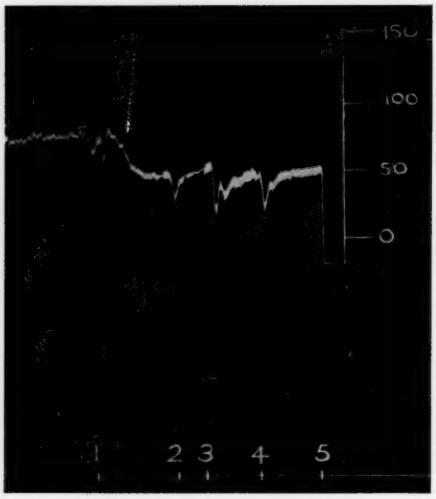

FIG. 622.—Showing the effect of injection into a test animal of material derived from superior mesenteric strangulation experiment. Sig. 1, 2 c.c. plasma after 1 hour; Sig. 3, 0·5 c.c. urine after 1 hour; Sig. 4, 2 c.c. peritoneal fluid after 1 hour; Sig. 5, 2 c.c. blood after 2 hours; Sig. 2, 2 c.c. dialysate from urine of another cat with non-viable strangulation of 21 hours' duration.

It was found that 5 c.c. of urine from a cat with superior mesenteric strangulation of one hour's duration was markedly depressor (*Fig.* 622, Sig. 3), as were also the gut blood (Sig. 1) and peritoneal fluid (Sig. 4) from the same case. Two c.c. of the urine from an animal with

a non-viable black loop at twenty and a half hours was dialysed against normal saline; 2 c.c. of this dialysate also gave a depressor reaction (*Fig.* 622, Sig. 2). This tracing also shows the cardiac death produced by the injection of 3 c.c. of blood after two hours' strangulation (Sig. 5). Following the injection of depressor substance into normal cats we found that the urine of the injected animal gave a depressor reaction, whereas normal urine was pressor. The identification of this substance in the urine is proceeding. It is significant that it is readily dialysable, as has already been demonstrated in the case of depressor substance elsewhere. It is not present in the urine before strangulation, nor can it be demonstrated in appreciable amount in normal animals before the intravenous injection of depressor substance. We are therefore tempted to regard it as an excretion.

Human Material

Some preliminary investigations have been conducted on material obtained from human cases of intestinal strangulation. In 3 instances in which a sample of peritoneal fluid was obtained no marked depressor effect was observed. In these cases the gut was viable and the strangulation of short duration. In 5 cases the urine was also examined with a view to determining whether excretion of a depressor substance had occurred. As mentioned previously, Abelous and Bardier[51] identified the existence of two components in normal urine. One component present in large amounts produces a marked pressor effect, so that the injections of small quantities of normal urine intravenously raise the blood-pressure, thereby masking the effects of a second component normally present in very small quantities, which, when extracted from large volumes of concentrated urine, has a depressor action. Bauer[50] showed that a blood-pressure-reducing principle present in very small amounts in normal urine was capable of being dialysed, and differed significantly in its properties from acetylcholine, adenylic acid, and histamine. He suggested that it possibly corresponded with the depressor substance not yet chemically defined which Euler and Gaddum had extracted from various tissues, including especially the smooth muscles of the intestinal wall. The similarity of these characteristics to those already noted in the case of depressor substance arising in the bowel wall, attracted our attention. It seemed justifiable to investigate the possibility that, under conditions of venous strangulation of the intestine, an increased amount of this depressor substance might be released into the circulation and give rise to an increased concentration in the urine. We have investigated the effects of urine from 5 cases of intestinal strangulation. In all of these a marked depressor effect was demonstrable on injecting a few cubic centimetres of urine. In such small doses urine from normal individuals was without effect on the blood-pressure, or alternatively produced a slight pressor reaction. The depressor substance was found to be readily dialysable. (*Fig.* 623.)

This tracing illustrates the injection of small quantities of the dialysate prepared from the urine of a patient with a viable strangulation of five hours' duration involving 18 in. of small intestine. In contrast to the similar dialysate from normal human urine which is faintly pressor, a marked depressor reaction is seen in the samples both before and after operation. It will be noted that the depressor effect is not precisely comparable in the samples before and after operation. This difference has been constant in all specimens examined, and may be due to differences

in concentration. In one case we have shown that this depressor effect, present at the time of operation, was no longer demonstrable on the seventh post-operative day. This fact, therefore, would seem to substantiate the hypothesis that the

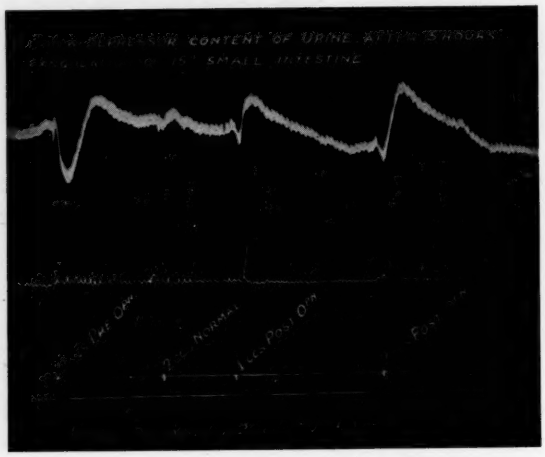

FIG. 623.—Effect of injection of dialysate of urine from human case of intestinal strangulation. Urine dialysed 20 c.c. v. 50 c.c. Sig. 1, 2 c.c. dialysate before relief of strangulation; Sig. 2, 2 c.c. dialysate of normal human urine; Sig. 3, 1 c.c. dialysate of urine after operation; Sig. 4, 2 c.c. dialysate of urine after operation.

appearance of this principle in marked concentration in the urine is definitely related to the occurrence of strangulation of the intestine. Our preliminary results are summarized in *Table XI.*

Table XI

CASE	NATURE OF STRANGULATION	EFFECT ON BLOOD-PRESSURE	
		Urine	Peritoneal Fluid
1	Femoral hernia—8-in. strangulation, 1 in. doubtful viability. Invaginated, died	Marked depressor ..	Not tested
2	Femoral hernia—5 in. viable small intestine	Not tested	Pressor
3	Femoral hernia—18 in. viable small intestine	Depressor before and after operation	Not tested
4	Internal band strangulation—12 in. viable small intestine	After operation depressor	Pressor ?
5	Inguinal hernia—knuckle of viable gut	Before and after operation depressor—7th day no effect	Pressor

DISCUSSION

From the introductory survey it will be seen that in order to account for the early onset of circulatory collapse which is so marked a feature of severe cases of intestinal strangulation and differentiates this condition from simple obstruction,

a mechanism which is capable of acting shortly after the onset must be sought. It has been shown that owing to their long latency, the factors disclosed by research into simple obstruction are inadequate in this respect, although they may be contributory in the later stages. Hypochloræmia alone can play no part in producing death, since death takes place in the presence of almost normal blood-chlorides. Loss of circulating fluid has been shown to play no more than an accessory rôle, whatever the length of the loop involved. There remain nervous or chemical factors. With the former we do not attempt to deal in this paper. With regard to the latter, in severe strangulation a depressor substance has been demonstrated in the wall of the intestinal tract within thirty minutes to one hour of the onset of venous obstruction and appears in the peritoneal fluid within one hour and in the thoracic duct within forty to seventy minutes. The first condition which any lethal factor must fulfil in order to account for the rapid collapse seen in severe cases, that of potential rapidity of action, is, therefore, satisfied. The second is that it should be capable of producing in the normal animal effects similar to those seen in the animal whose gut has been strangulated. In this early report of our first year's investigation we cannot advance complete evidence on this point. The pharmacodynamic action of the substance and the precise effects of continued slow absorption either alone or together with ancillary factors is the subject of further research. It has been shown, however, that the peritoneal absorption of 35 c.c. of the exudate from a black loop at sixteen and a half hours fulfilled this condition by causing death within three and a half hours in the normal animal. Since there is such complete similarity between the reactions of depressor substance in peritoneal fluid, gut blood, and elsewhere, it may be inferred that its slow continuous absorption from any site will produce a similar effect. That repeated or large doses do produce a permanent depression of blood-pressure comparable with the clinical picture of strangulation is a characteristic of all our reactions. That accessory factors other than the presence of depressor substance exist we unhesitatingly agree. Foremost among these is distension, and especially, in our opinion, rhythmic alteration in the degree of distension, as occurs in the course of peristalsis. Whether the significance of distension lies in a nervous mechanism, its influence on absorption, or the further production of depressor substance, is as yet not completely established. Our present bias is towards the last two factors. To a lesser degree loss of fluid may contribute to the circulatory collapse, but in no case, even in very long loops, can dehydration alone be sufficient to account for death.

The origin of depressor substance from the tissues of the gut wall as the result of changes within them, shortly after the onset of strangulation, has been demonstrated, and accords with physiological possibilities. This origin serves to differentiate it from any form of toxæmia previously described. Toxæmia has been attributed by most workers to changes occurring inside the gut lumen. Whipple, it is true, held strongly that the toxin of simple obstruction originated from the mucosa after twenty-four hours, but the fact that the depressor substance investigated by us is readily dialysable serves as a point of differentiation from his toxic proteose. Holt believed that a toxin arose in the later stages of strangulation from bacterial action on the gut wall.

While we do not dispute that bacterial invasion of devitalized tissue and subsequent proteolytic action may produce further toxic substances from the tissues of the gut wall in the latter stages of strangulation where the viability of the gut is

impaired, the early origin demonstrated for depressor substance, when the gut lumen was washed through and contained only saline, in our view definitely precludes a bacterial origin. In the light of this factor it is necessary to review the results of previous experimental study in relation to the early collapse of intestinal strangulation. Although the peritoneal absorption of toxic substances was recognized by Foster and Hausler in the late stages of short-loop strangulation, the initial collapse in severe cases of the long-loop type was ascribed to 'shock', although no precise evidence as to its causation was advanced. Holt, in repeating these experiments, again differentiates between the cause of death in short and long loops, in the latter case believing that death was entirely due to loss of fluid from the circulation. By cross-circulation experiments this latter view has been shown to be untenable. As will be seen from his method of forming loops by individual venous ligature with patent lymphatics, it is precisely in this type of case that lymphatic absorption of toxic substances most readily occurs. We therefore hold on the basis of present evidence that the early collapse of severe cases of intestinal strangulation may be attributed to toxæmia from the absorption of depressor substance. Distension is undoubtedly an important contributory factor. Loss of fluid plays a variable and entirely accessory rôle, becoming more important the greater the length of the gut affected. Hypochloræmia and bacterial toxæmia are without significance at this stage. In the later stages, if the gut viability is impaired, this toxæmia may be reinforced by a further process. Bacterial invasion of the devitalized tissue may, as Holt suggests, produce further toxins by proteolytic action.

A study of our results shows that there is a rough correspondence between the survival period and the length of loop involved. We do not on this account think that it is justifiable to segregate cases of strangulation into arbitrary groups classified according to the length of gut involved, with a *different* pathogenic factor for each group. Where longer loops are affected, fluid loss of course plays a more important part, but with such loops there is necessarily a larger area for the production of toxic substances. In very short loops, less toxin being produced, life may be prolonged sufficiently to allow the latent period for the development of the toxæmia of the associated simple obstruction to be passed, and this may contribute to the collapse in the later stages. In general it may be said that the significance of the length of loop would seem to lie in the increased or decreased potential area for the formation of toxic substances. We have shown that of greater importance than the length of loop involved is the severity of the strangulation. Admittedly the combination of length with severity as in superior mesenteric strangulation gives the most rapidly fatal result, but with smaller loops of equal length, the severity is the determining factor, which accords with clinical experience. It has been shown that the more severe strangulation is accompanied by more rapid appearance of depressor substance in the peritoneal fluid and thoracic duct, and in certain cases by greater concentration in the gut blood.

It has been shown that in those cases where, as is usual in the human, the mesenteric lymphatics are compressed, the ultimate route of absorption is by way of the peritoneal cavity, and that this only contains depressor substance when the strangulation is severe. Our results show that dye-stuffs will be absorbed into the circulation via the lymphatics, if introduced into the tunica vaginalis or the peritoneal cavity. There seems no justification, therefore, for the view sometimes expressed that an enterocœle in a closed hernia sac should be regarded as an extraperitoneal

structure, and, therefore, comparable to a simple obstruction. The lesser mortality shown statistically in these cases is far more probably due to early diagnosis. The significance of absorption via the thoracic duct from the peritoneum is seen from the fact that any substance delivered by this route reaches the circulation directly without the possibility of detoxication by the liver, and so may produce a progressive action despite gradual elimination by the kidney. The similarity of the urinary reactions of strangulated and injected cats and those of human patients with known strangulation suggests that the same or some closely allied depressor substance is present in the human. If this is so, certain interesting considerations arise. What rôle, if any, does renal function play in determining the fatal outcome of a case of strangulation? Does treatment by saline therapy and infusion, which, as already shown, is not directed to the cause of the collapse, produce the beneficial effect sometimes observed through its associated diuretic action? Finally, what is the significance of the release into the circulation of that portion of depressor substance which is dammed up in the veins and lymphatics of the strangulated loop? In a patient who is in good condition the release of this substance into the circulation must be well tolerated; but in the toxic case the sudden release into the circulation of a large dose of depressor substance, which, as already shown, causes a profound and permanent lowering of the blood-pressure, may be sufficient to determine a fatal outcome. Should we, therefore, in these cases make an attempt to remove the toxic substance in the peritoneal fluid and gut wall, and, having regard more to the viability of the patient than to the viability of the gut, should excision of the affected segment be performed?

Admittedly the smallest operation consistent with the pathological condition of the gut is held to give the smallest mortality. But many observers believe that once distension and toxæmia have set in, only resection can save the patient. The statistical figures give no information which truly illustrates this point, but it would be interesting to compare the results of resection and relief *in comparable cases of equal severity*. Alternatively, there remains the hope that further research may reveal some method of combating the effects of the toxin. These, together with the other points mentioned in the text, are subjects of continued investigation.

SUMMARY

1. The importance of the factor of venous strangulation as opposed to simple obstruction is emphasized.

2. Dehydration and hypochloræmia are shown to be inadequate to account for the fatal outcome of strangulation even in very long loops.

3. The production of a toxic substance from changes in the tissues of the gut wall within one hour of the onset of strangulation is demonstrated.

4. This substance produces a characteristic and persistent depression of blood-pressure which corresponds to the clinical picture of strangulation.

5. Where the mesenteric veins and lymphatics are compressed, this substance is ultimately absorbed through the peritoneal fluid and thoracic duct.

6. The relief of experimentally produced strangulation results in deterioration of the animal's condition, presumably owing to the flooding of the circulation with the accumulated toxin which is shown to appear in the mesenteric lymphatics and veins after a period of strangulation.

7. This toxic substance has been recognized both in the urine of experimental animals and human patients with known strangulation.

In conclusion we would like to take this opportunity of expressing our thanks to Mr. S. P. Steward and the members of the Technical Staff of the Royal College of Surgeons Research Department and of the Buckston Browne Farm for their expert assistance at all stages of this investigation.

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SHORT NOTES OF RARE OR OBSCURE CASES

A CASE OF HAIR-BALL OF THE STOMACH AND DUODENUM IN A CHILD OF $3\frac{1}{2}$ YEARS

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A BOY, aged $3\frac{1}{2}$ years, was admitted to the Royal Aberdeen Hospital for Sick Children under my care on June 7, 1934.

HISTORY.—His mother stated that he had been a healthy child till November, 1933, when he had an attack of sickness and vomiting which lasted a week but subsided after a dose of castor oil; from then onwards he appeared fretty and

FIG. 624.—Radiograph showing filling defect in stomach— and third part of duodenum.

often woke up crying. Five months later he was seized with acute abdominal pain and diarrhœa, and received treatment in hospital for anæmia and an enlarged spleen. His condition improved, only to relapse again in June, when he was treated in another hospital without success. He had never been jaundiced. His family history was negative.

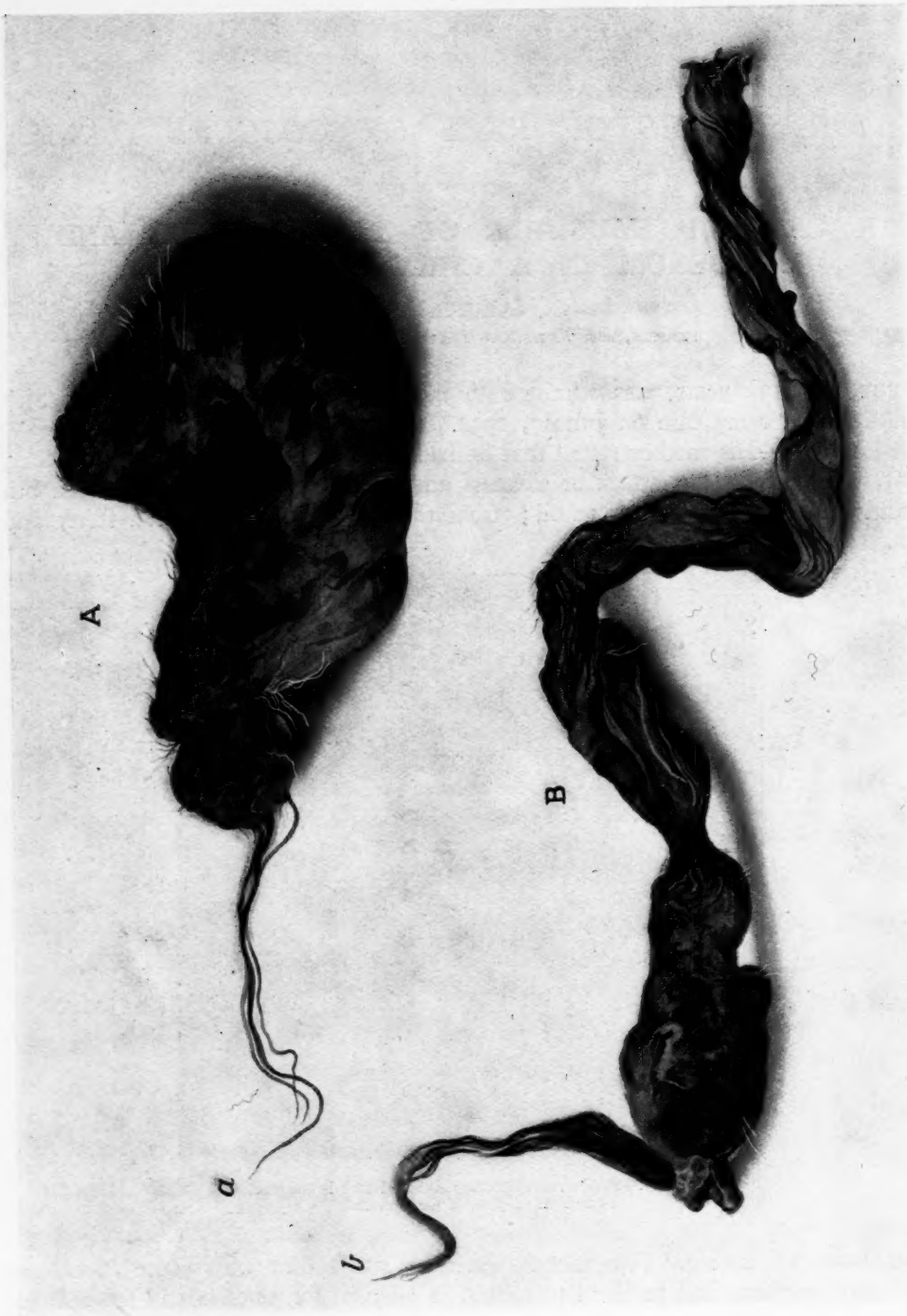

FIG. 625.—A, Hair-ball removed at operation; B, Hair-ball passed per rectum three days after operation.

ON EXAMINATION.—The child looked thin and dehydrated, fretty, and restless. His temperature was 98°, pulse 128, respirations 28. His tongue was furred and his breath foul. The abdomen was distended. In the epigastric angle and beneath the left costal margin a large solid mass could be felt which moved with respiration and was not tender. In shape it suggested a cast of the entire stomach and appeared obviously gastric in origin. The liver, spleen, and kidneys were not palpable. The stools were pale and clay-coloured. Bile was present in the urine, which was otherwise normal. The vomit consisted of altered food and bile.

Upon radiographic examination, shadows suggestive of beads were seen in the region of the fundus of the stomach, and it appeared unlikely that such would remain in a child's stomach unless entangled in a foreign substance. A hair-ball of the stomach was suspected. On questioning the mother, it was found that she had seen her boy picking wool off his jersey and eating it; he also plucked wool out of the mats and pulled out his own hair. A barium meal showed a large filling defect in the stomach (*Fig.* 624).

An attempt was made to improve the boy's health with small feeds, salines, etc., but his condition rapidly deteriorated; the vomiting became more persistent and the child more and more dehydrated. Although he appeared a serious risk, I decided to delay operation no longer.

OPERATION (June 17).—Gas and oxygen anæsthesia. The abdomen was opened through a right paramedian incision and the stomach was found almost completely filled with a soft, semi-solid substance which extended from the cardiac end through the pylorus. The surrounding viscera were packed off with moist gauze strips and the stomach was opened by an incision at right angles to the greater curvature. A large, greenish mass (*Fig.* 625A) approximately 6 in. × 3½ in. composed of hair, wool, and string was delivered with difficulty from the stomach. It could not be completely withdrawn on account of a few strands of string which extended through the pylorus into the duodenum. These were divided, the hair-ball extracted, and the incision in the stomach wall was closed with two layers of sutures.

SUBSEQUENT HISTORY.—Three days after operation the child passed per rectum a long mass (*Fig.* 625B) 14 in. in length and about 3½ in. in circumference composed of the same substances as the hair-ball removed from the stomach. From its length, the second hair-ball must have extended through the duodenum to beyond the duodeno-jejunal junction, and had prevented the extraction of the gastric mass until the communicating strands were divided as they passed through the pylorus.

Convalescence was stormy. A few days after operation crepitations could be heard at both bases, and four days later dehiscence took place. Several feet of bowel prolapsed through the wound, and a ragged tear was found in the jejunum from which intestinal contents exuded. The patient was transferred to the operation theatre without delay, when the jejunal opening was repaired, the intestine returned to the abdomen, and the abdominal wall closed with through-and-through sutures of silkworm gut. He recovered and is now in excellent health. A barium meal shows the stomach to be normal.

HÆMANGIOMA OF THE EPIDIDYMIS

By A. L. D'ABREU

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Cystic and inflammatory swellings of the epididymis are common. Solid tumours are extremely rare, and fall into three groups (Rubaschow[1]): (1) Heterotopic tumours arising from embryonal rests; (2) Carcinoma arising from specific epididymal tissue; (3) Tumours arising from local neutral tissues such as sarcoma, fibroma, and lipoma. The case of hæmangioma recorded here must be placed in the third group.

E. P., aged 28, was admitted to the Surgical Unit of the Welsh National School of Medicine on Aug. 31, 1935, complaining of a swelling in the right side of the scrotum of six weeks' duration. On awakening one morning he felt a little pain and was astonished to find a swelling there. He was emphatic that there had been no trauma. At the end of ten days the pain had disappeared and the swelling had decreased in size. There had been no urethral discharge nor any difficulty in micturition.

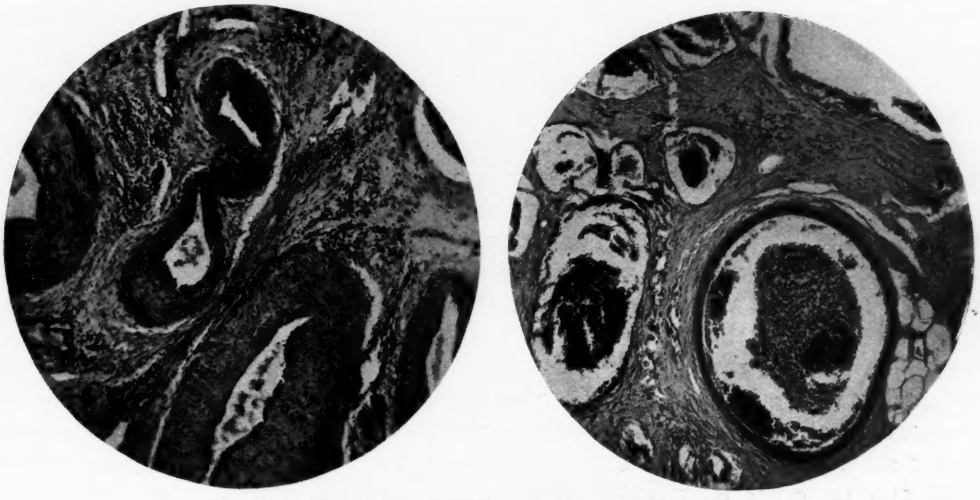
Fig. 626.—Appearances seen after the testis had been bisected. The epididymis is replaced by spongy hæmangiomatous tissue. The body of the testis is compressed and there is blood-clot in the tunica vaginalis. The arrow indicates the line of division between the angioma above and the compressed testicular tissue below.

On Examination.—When he presented himself for examination six weeks later, a well-developed and healthy man, the right testicle was twice the size of the left, which was normal in all respects. The epididymis appeared to be fused with the body of the testis for no separation of the two structures could be appreciated by digital examination. The skin of the scrotum in front of the testis was slightly adherent to an underlying mass, which was firm in consistency and felt moderately heavy to the supporting hand. Testicular sensation was absent. The constituents of the cord were normal on palpation. These findings betokened a syphilitic origin, but the Wassermann reaction was negative.

A diagnosis of malignant disease of the testicle was therefore made because of the painless enlargement, the skin adherence, and the absence of signs of infective epididymo-orchitis. To exclude the possibility of a tumour containing chorion-epitheliomatous tissue, fresh morning urine was sent to the Pregnancy Diagnosis Station of the Pathological Department of the Welsh National School of Medicine, where Dr. J. Gough carried out the Friedman test. He reported that the reactions in the rabbits tested were negative and that chorionepithelioma could be safely excluded from the diagnosis.

OPERATION.—The cord having been mobilized and divided at the internal abdominal ring, the testicle with the adherent skin and the entire tunica vaginalis was removed in one piece. Convalescence was uneventful and the wound healed by first intention.

PATHOLOGY OF THE REMOVED TISSUE (*Fig.* 626).—The tunica vaginalis contained recent blood-clot in the process of organization: the skin adherence noted before the operation was due to this process, the subcutaneous tissues and the blood-clot in the tunica vaginalis having undergone fibrotic organization. The origin of this blood was soon obvious, for the whole of the epididymis appeared to be replaced by thin-walled angiomatous tissue which had given way in several places with extravasation of blood. Microscopically (*Figs.* 627, 628) this tissue was characteristically that of a cavernous angioma, and only a few epididymal tubules



FIGS. 627, 628.—The microscopical appearances of the tumour.

were seen. The body of the testis was compressed from above by the angiomatous mass. No evidence either macroscopically or microscopically of a malignant neoplasm could be found.

I can find details of only one other case, that recorded by J. P. Hosford.[2] His patient was a youth of 15 who complained of a rapid enlargement of the right testis following a blow. On examination the organ was enlarged to twice its normal size, firm in consistency, and not tender; the epididymis could not be felt. Five years after orchidectomy the patient was alive and well. An examination of the specimen revealed the normal body of the testis compressed from above by a mass a little larger than itself consisting of a cavernous angioma which replaced the epididymis; no elements could be found to suggest a teratomatous origin.

I am indebted to Dr. J. Gough for his kindness in carrying out the Friedman test.

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A CASE OF MESENTERIC TUMOUR

By A. WALLIS KENDALL
ASSISTANT SURGEON TO KING'S COLLEGE HOSPITAL

E. F. P., male, aged 49, a shop-assistant, was admitted to hospital under my care on Sept. 9, 1935.

History.—For the past nine years the patient had complained of indigestion, which had become worse during the last six months. After meals he experienced a sense of fullness and also a dull aching sensation in the left iliac region. There was no vomiting. He stated that he was becoming constipated but that he was able to correct this by taking liquid paraffin. He had noticed no disturbance of micturition during the day, but latterly had been obliged to get out of bed once each night. He was unaware of having an abdominal swelling previous to having been examined. He stressed the fact that none of his symptoms was in any way severe.

On Examination.—A mass could be seen to cause some prominence of the lower central abdomen, extending from the symphysis pubis to the level of the umbilicus. The swelling was roughly oval in shape, but there was a prominent boss, the size of a tennis-ball, projecting from the upper right pole. The upper and lateral borders were rounded and well-defined. The lower border could not be made out, but it was obvious from the fact that it was beyond the reach of the examining finger in the rectum that the swelling could only extend a short distance below the level of the symphysis pubis. Apart from an indefinite suggestion of division into large lobules the surface was smooth. The consistency was firm and fluctuation not obtained. The swelling could be moved easily from side to side over a short distance. Palpation was quite painless. There were no signs of free fluid and no other abnormality discoverable in the abdomen. An intravenous pyelogram showed a normal urinary tract. A blood-count showed 5,500,000 red blood-corpuscles and 20,000 white blood-corpuscles per c.mm.; hæmoglobin 92 per cent; colour index 0·83.

Operation.—On Sept. 17 the peritoneal cavity was opened through a mid-line lower abdominal incision. There was no free fluid present in the peritoneal cavity. The tumour was found to be attached above to the mesentery of the small intestine in close proximity to the gut, and below deeply in the pelvis to the posterior aspect of the bladder on the left side. In each case a pedicle was present, the upper being shorter, wider, and considerably more vascular than the lower. The bulk of the tumour was delivered through the abdominal incision with the attached loop of small intestine. The pedicle ($\frac{1}{2}$ in. long $\times$ $1\frac{1}{2}$ in. wide) to the mesentery, whose vessels were several times their normal size in this situation, was ligatured and cut across and the small intestine set free from the tumour. The blood-supply to the intestine did not appear to be interfered with. The lower pedicle ($1\frac{1}{2}$ in. long $\times$ $\frac{3}{4}$ in. wide), which had only one or two small vessels in it, was now also ligatured and cut across. The tumour having been removed, the patient was placed in the Trendelenburg position and the intestines were packed off. It was then seen that in addition to the stump of the lower pedicle there was also a small raw area in a corresponding position on the right posterior aspect of

the bladder where adhesion to the tumour had occurred. Both the pedicle stump and this area were buried and the abdomen closed.

The patient made an uneventful recovery and was discharged from hospital on the fourteenth day.

FIG 629.—Photograph of tumour after removal. (× $\frac{2}{5}$.)

FIG. 630.—Photograph of tumour after section. (× $\frac{2}{5}$.)

It will be seen from the photographs (*Figs.* 629, 630) that the tumour, which weighed 2 lb. 9½ oz., is partly cystic but is for the greater part solid. There is some attempt at fibrous encapsulation of portions of the mass, particularly noticeable in the projecting upper pole. Except at the points of attachment the tumour is covered by peritoneum, underneath which can be seen large thin-walled veins.

On section the cystic, hæmorrhagic, and yellowish degenerative areas gave an appearance not unlike that of an advanced hypernephroma (*Fig.* 630).

Histological examination (*Fig.* 631) of different parts of the tumour was made by Dr. Creed, to whom I am indebted for the following report: "The section shows a sarcomatous growth which varies in appearance in different parts of the tumour. In the solid parts the growth is spindle-celled and the cells are arranged in whorls. In the more cystic part there is more production of collagen and the tumour is more fibrosarcomatous, with trabeculæ of dense fibrous tissue. The tumour is very vascular, but the vessels are well-formed, and show a good deal of hyaline degeneration."

FIG. 631.—Histological appearance of tumour. (× 200.)

Comment.—Though of rare occurrence, a sufficient number of mesenteric tumours have been described in the literature to show that considerable variation in their pathological nature exists. About half are malignant in type, and of these only one-third are removable at operation. Particular reference must be made to the article by Phillips,[1] who publishes a series of 2 solid mesenteric and 4 retroperitoneal tumours, and suggests, on strong pathological grounds, that these two groups be classed together as paramesenteric tumours. In practically every recorded case the slightness of the symptoms in the presence of a large tumour is noted. In spite of the pathological nature and size of the growth described here it is interesting to note the absence of technical difficulties in the way of its removal, and also the apparent freedom from metastasis.

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A LARGE LEIOMYOMA OF THE PROSTATE

By T. W. MIMPRISS
CHIEF ASSISTANT, SURGICAL UNIT, ST. THOMAS'S HOSPITAL

A LABOURER, aged 60, was admitted to hospital with acute retention of urine. During the previous year he had suffered from progressive frequency of micturition; in the month before admission he had had a daily frequency of nine and nocturnal frequency of five or six times. The stream was poor and he experienced difficulty in beginning the act. There was a history of one recent attack of acute retention, relieved by catheterization. No other urinary symptoms had been noted.

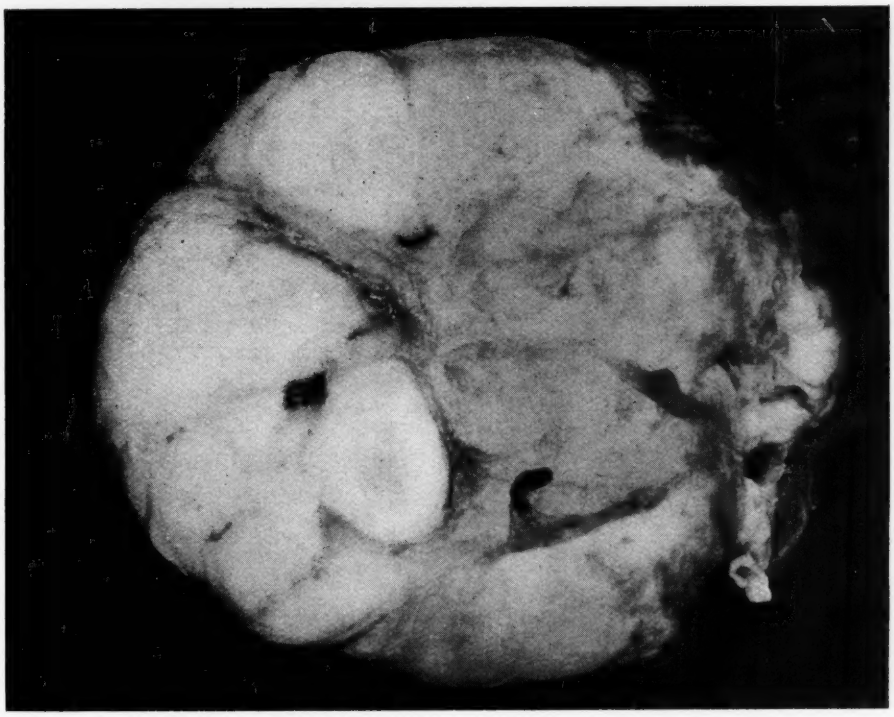

FIG. 632.—Leiomyoma of prostate. (× ⅜.)

ON EXAMINATION.—The patient was a healthy man whose bladder extended to the umbilicus. Rectal examination showed a very large prostate, only the lower part of which could be examined by the finger. The gland was smooth and firm, suggesting the common type of prostatic enlargement. Gradual decompression by means of an indwelling catheter was carried out over a period of twenty-four hours. No abnormality was detected in the respiratory and cardiovascular system, the blood-pressure was 150/90, the blood-urea 32 mgrm. per 100 c.c., and the urine free from infection. Indwelling catheter drainage with twelve-hourly bladder washes was carried out for ten days. In view of his good condition a one-stage suprapubic prostatectomy was then performed.

OPERATION.—Under ether anæsthesia the prostate could be palpated through the abdominal wall. On opening the bladder an enormous prostate was found. It surrounded the urethra and appeared to be an extreme example of the ordinary type of prostatic enlargement. Owing to its size it was impacted between the body of the pubis in front and the sacrum behind. The tumour could be enucleated from its capsule with ease, but great difficulty was experienced in reaching the lower part of the tumour, as it filled the pelvis almost completely. Enucleation therefore involved considerable manipulation and some degree of hæmorrhage.

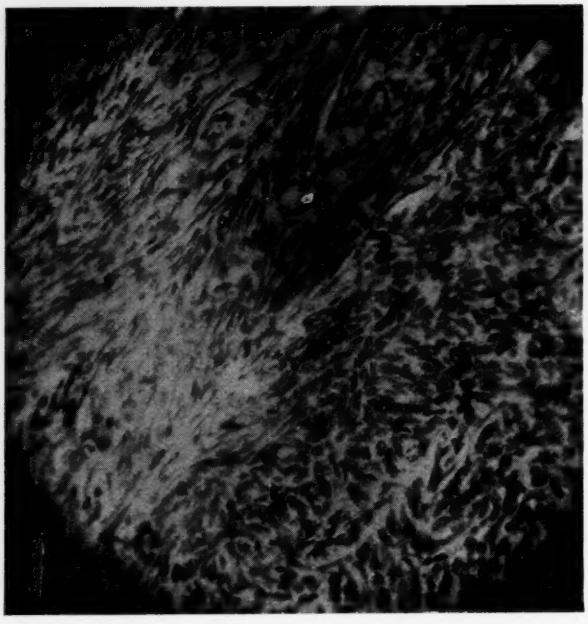

FIG. 633.—Low-power view of section of tumour.

The patient was much shocked at the end of the operation and died twenty hours later.

PATHOLOGY.—The specimen (*Fig.* 632) weighed 19 oz. and measured 5·6 × 4·4 × 2 in. Macroscopically it was of the same structure throughout. Numerous sections (*Figs.* 633, 634) of the gland submitted to microscopy showed the structure of a leiomyoma; no section showed any trace of glandular tissue.

Discussion.—It is recognized that small leiomyomatous nodules are found comparatively frequently in enlarged prostates. Large leiomyomata are rare.

Dial and Halpert[1] in 1933 reviewed the literature on this subject and divided the cases into two groups, depending on whether the tumour caused disturbances of micturition or of defæcation. The case recorded here comes into the former group.

Patch and Rhea[2] have recently reported a case of this type, together with a review of the literature. Including their own case, they find only 11 cases of large leiomyomata reported. They also point out that in some of the recorded cases

the tumour was attached to the prostate by a pedicle, as occurred in Hinman and Sullivan's[3] first case, or was situated in a position remote from the urethra.

In the case recorded here the tumour was peri-urethral and projected into the bladder. Before the specimen was enucleated it resembled exactly the usual type of prostatic enlargement. The weight of the specimen (19 oz.) is remarkable. A search of the reports of large prostatic tumours of any type suggests that it is the second largest recorded. Neupert[4] reports a retroperitoneal tumour weighing $7\frac{1}{2}$ lb., which was considered to be a large leiomyoma in connection with the prostate. The case report, however, is not very explicit. Apart from this, the

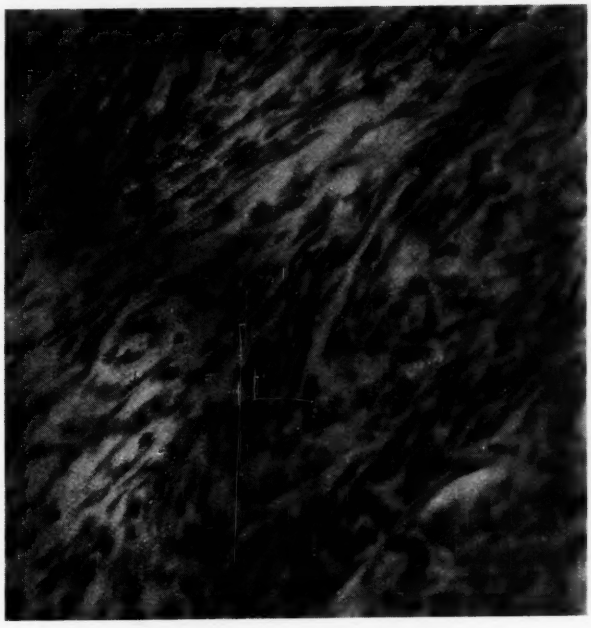

FIG. 634.—High-power view of section of tumour.

largest recorded tumours I can find are those of Fullerton[5] (17 oz.) and Freyer[6] ($16\frac{3}{4}$ oz.). Both of these tumours were of the ordinary type of prostatic enlargement.

I wish to thank Professor L. S. Dudgeon for his advice and pathological report on the specimen, and Mr. W. H. C. Romanis for permission to publish the case.

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[2] PATCH, F. S., and RHEA, L. J., *Brit. Jour. Urol.*, 1935, vii, 213.
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A LIPOMA OF THE COLON

By KEITH ROSS

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LIPOMATA of the colon are so rare that the clinical notes of a new case may be of sufficient interest to warrant their publication.

Mrs. M., aged 40, had suffered for some months from flatulence and discomfort in the epigastrium. The discomfort usually occurred soon after meals and was relieved to some extent by regulation of the diet on ' ulcer ' lines. Relief, however, was incomplete, and further examination disclosed a mobile rounded tumour which could be felt in the left epigastrium, only when the patient was lying on the right side. There was no disturbance of the bowels. Examination by a radiologist after a barium meal and enema showed no lesion of the gastro-intestinal tract.

Laparotomy revealed the ascending colon to be very considerably enlarged and a tumour was felt within the lumen of the colon at its hepatic flexure. The

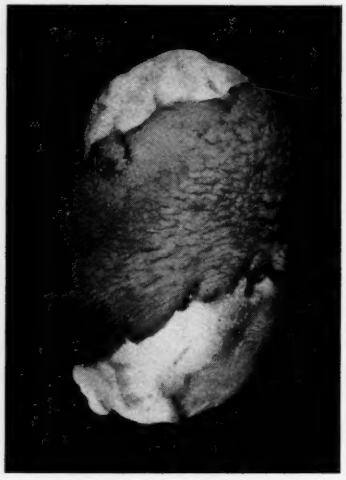

FIG. 635.—Lipoma ulcerating through mucous membrane of colon.

tumour could be ' milked ' along to the left half of the transverse colon in one direction, and to the cæcum in the other, without causing any invagination or dimpling of the wall of the bowel. The tumour was then fixed in the cæcum and the latter delivered through a gridiron incision in the right iliac fossa. On opening the cæcum, the tumour was found to be a large oval lipoma ($3\frac{1}{2} \times 2$ in.) arising in the submucous tissue, pushing the mucous membrane before it, and ulcerating through the mucous membrane. The mobility of the tumour was due to its having a long pedicle of loose mucous membrane, and its removal was easily accomplished by cutting through the mucosa at its reflection on to the mass. The mucosa was sutured, and as soon as it was let go it sprang sharply back up the ascending colon. The cæcum and abdominal wounds were closed and the patient had an uneventful convalescence.

The tumour, which is shown in *Fig.* 635, has been carefully examined and found to consist of fatty tissue only.

LOCAL PLASTIC PERITONITIS, POSSIBLY DUE TO INCARCERATION OF INTESTINE IN A HERNIA, CAUSING INTESTINAL OBSTRUCTION

By KEITH ROSS

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CHRONIC adhesive peritonitis may occur as a local or a generalized condition and in varying grades of severity. In many cases it would appear that the inflammatory process originates in the female pelvic organs and spreads thence to the bowel. In others, such as the one reported here, the etiology is more obscure.

J. W., a male aged 58, was admitted to hospital with a three days' history of severe pain in the abdomen, vomiting, and absolute constipation. Previously, apart from a mild and non-progressive constipation, he had always been healthy.

FIG. 636.—Mass excised: now collapsed, but tensely swollen at operation. ($\times \frac{1}{2}$.)

He had never had an operation or suffered from any disease such as typhoid fever or dysentery. For many years he had had a right inguinal hernia and this had come down since the onset of vomiting. It was easily reduced and found to have a widely patent neck.

OPERATION.—Immediate laparotomy disclosed a large tumour consisting of a mass of ballooned and inextricably fused coils of ileum lying quite discretely below the right lobe of the liver. A grossly hypertrophied loop of ileum ran to the mass and a correspondingly small one from it. There were no adhesions to

surrounding structures, and a rapid survey revealed no other morbid process within the abdomen. The mass was excised and the ileum re-united by a side-to-side anastomosis. Convalescence was uneventful.

After operation the blood was found to give a negative reaction to the Wassermann test; X-ray examination of the chest disclosed no evidence of pulmonary tuberculosis or any thickening of the pleura; there was no evidence of renal disease.

PATHOLOGICAL FINDINGS.—The mass excised is shown in *Fig.* 636 and consists of numerous loops of ileum firmly bound together by fibrous adhesions. At the junction of the afferent loop of ileum and the mass there is a dense white scar (not very well displayed in the illustration) and the appearance of constriction. Water pumped into the mass from either end passes only very slowly through it. On dissection the gut is found to loop acutely back on itself in maze-like fashion, with

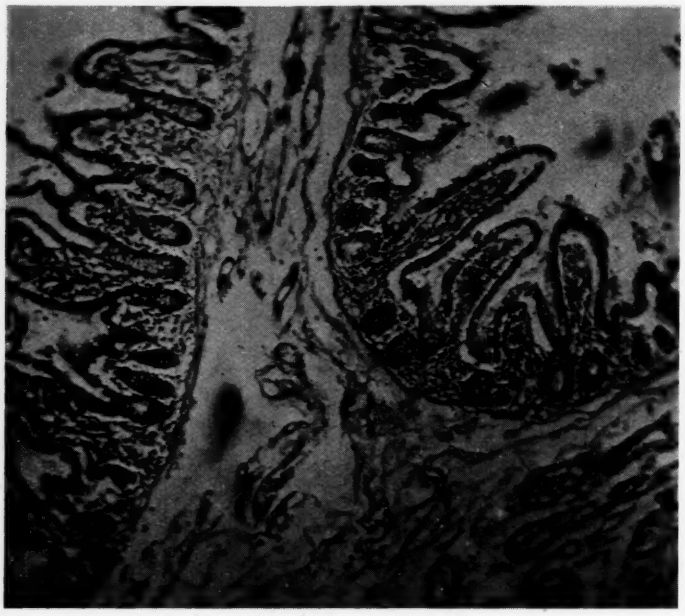

FIG. 637.—Microscopic view of two fused loops.

constriction of the lumen in very many places, but there is no pathological process evident other than fibrosis. Microscopic examination of the affected loops discloses thickening of the tunica serosa (*Fig.* 637).

Comment.—The etiology of the pathological condition in this case is obscure. So far as can be ascertained there had never been any lesion or disease of any nature whatsoever of any of the abdominal organs, nor was there any evidence of syphilis or tuberculosis. There was, however, a wide-necked hernia and this had frequently contained bowel. Bowel which has remained for any length of time in a hernial sac not infrequently shows evidence of chronic peritonitis, and it appears reasonable to suggest that in this case the condition had originated in one or more loops of ileum that had been retained for some time in the hernial sac; later when the gut returned to the abdominal cavity the pathological process continued and extended. This explanation is purely hypothetical, but on the evidence available it appears to be the most reasonable.

TRANSFIXION OF THE AORTA

By KENNETH EDMUNDSON
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THE following case would appear worthy of record because the patient was still alive thirteen hours after he had received an apparently fatal wound, and because the inflicting weapon acted as a preventive to further damage. The facts are as follows :—

A male African was brought into hospital at 1 p.m. by motor-car from a distance of fifteen miles. He had been wounded by an arrow, being shot at close quarters the evening before at 11 p.m. He was in no obvious distress on arrival at hospital and walked unaided from the car to the operating-theatre, a distance of some fifty yards.

ON EXAMINATION.—The iron shaft of an arrow-head protruded 1 in. from the sternum. It was about ½ in. to the right of the mid-line and opposite the second interspace. It protruded almost at right angles to the sternum, the direction being backwards and slightly upwards. This shaft was seen to vibrate rhythmically with the pulse, and on placing the finger lightly on the tip a transmitted thrill was felt. On auscultation near the shaft a loud musical murmur was audible, which was conducted up into the neck on the right side. The pulse on admission was 100. There had been very little external hæmorrhage up to this time, although obvious endeavours had been made to cut and pull the arrow-head out.

Soon after admission the wound began to bleed and the pulse quickened to 120. Morphia, ¼ gr., was given and firm pressure with swabs was applied over the wound. The hæmorrhage subsided. It was then decided to try to remove the arrow-head, as it was judged to be held under the innominate.

OPERATION.—Under slow chloroform and ether anæsthesia an incision was made in the mid-line of the sternum and the skin reflected. An oblong window, 2 in. by 1 in., was made in the sternum. The bone was lifted up over the arrow-shaft. The anterior mediastinum contained blood-clot, but no fresh bleeding was encountered on the removal of the window of bone. The lower barb of the arrow was first freed from tissue and then the upper, and the arrow was withdrawn. Its withdrawal was immediately followed by a very severe hæmorrhage. The patient died almost immediately.

POST-MORTEM EXAMINATION.—This was made as soon as possible after death, and showed the following facts. There was complete transfixion of the aorta by the arrow, which had entered the ascending portion on its convexity and had penetrated the posterior wall at the junction of the innominate and the aorta. The inner coat of the vessel showed superficial excoriations as if the point of the arrow had been moved to and fro in the lumen. There was no marked internal hæmorrhage, nor injury to the pleura or pericardium. The hæmatoma in the track of the arrow was inconsiderable and the hæmorrhage had not extended down into the posterior mediastinum.

Conclusion.—The arrow had pierced the sternum in such a manner that the two barbs just impinged on the under surface of the sternum, the point being fixed by the barbs in the aorta and the tissues posterior to it. There was thus formed

an immovable splint, which completely filled the incisions made and which the musculature of the vessel gripped and rendered efficacious. Removal of the plug formed by the arrow-head was followed by splitting of the muscle under the pressure and instant death.

The extraordinary feature of the case is the fact that the man had lived for over thirteen hours, and, in spite of the efforts of his friends to remove the arrow by cutting and pulling, survived until its removal by modern methods.

I have to thank the Director of Medical and Sanitary Services, Tanganyika Territory, for permission to publish this case.

REVIEWS AND NOTICES OF BOOKS

Post-graduate Surgery. Edited by RODNEY MAINGOT, F.R.C.S., Senior Surgeon to the Royal Waterloo Hospital, etc. With an Introduction by the Right Hon. LORD MOYNIHAN OF LEEDS, K.C.M.G., C.B., M.S., F.R.C.S. In three volumes. Royal 8vo. Vol. I: Pp. 1742 + xvi, with 846 illustrations. 1936. London: Medical Publications Ltd. £9 9s. the set, or £3 10s. per volume.

THIS great work on surgery has a very definite aim and scope. It presents the subject in a practical way, so that qualified surgeons may have a guide in their work and a book of reference in their reading. Basic knowledge of surgery is assumed, and the space is devoted to the description of special methods of investigation, special methods of treatment, including operative technique, and the management of cases both before and after operation.

The team of writers who have collaborated in producing this work is notable in two respects. In the first place it includes many young men—a fact which gives the best guarantee that the book shall be really up-to-date. And secondly it includes a large proportion of physicians (actually seven out of twenty in this first volume), which indicates that the treatise is no mere technical description of operative procedure, but a reasoned study of patients and of pathological processes. The present volume opens with an introduction by Lord Moynihan, who speaks of the needs of the post-graduate student and the high ideals of surgery.

Part I deals with anæsthesia (Langton Hewer). There is a clear account of anæsthetic methods, preference being given to gas–oxygen for inhalation and percaine for spinal anæsthesia. A cautious and critical account of premedication methods and of evipan is given.

Part II deals with the abdomen. This section occupies the greater part of the volume (nearly 1200 pages), but only includes the surgery of the alimentary tract, glands, and spleen. Preliminary chapters on the investigation of a case of dyspepsia are by Maingot, Sleigh Johnson, and Deville. The common diseases and the usual operative procedures on the stomach and duodenum are clearly described and well illustrated by Maingot, and then follows a special chapter on the Billroth I operation of gastrectomy by Finochietto (Buenos Aires) with a great number of detailed illustrations. Hurst writes on the medical treatment of gastric, duodenal, and anastomotic ulcer. In his opinion, if suitable treatment is given for a sufficient period, nearly every gastric ulcer eventually heals, as well as about 75 per cent of duodenal ulcers, and 50 per cent of the much more intractable anastomotic ulcers which follow gastric operations.

The chapters on the gall-bladder and bile-ducts are by Maingot, Moore, and Dickson Wright. In addition to good illustrations of diseased conditions and operative procedures there is a series of excellent diagrams of the mistakes and accidents which may occur in the performance of cholecystectomy.

The chapters on the liver are by Dickson Wright and Sleigh Johnson. Rupture of the liver is dismissed in half a page, but great space is devoted to liver function and jaundice. The diseases and injuries of the spleen are very fully discussed by Maingot, whilst the rôle of radiotherapy in splenic diseases is described by Levitt, and Egyptian splenomegaly by Stiven.

Appendicitis and its complications and treatment are described by Hamilton Bailey in a most lucid and helpful chapter. A very fair via media between the extreme views about treatment of the more-than-48-hours case is provided. This takes the form of laying down the rule of expectant treatment except under five circumstances. The whole article is very helpful in its clear advice and directions for treatment. In cases where pylephlebitis is feared, it is suggested that the ileocolic vein should be ligated before removing the appendix. We think that tuberculous peritonitis should be referred to in the differential diagnosis.

Wakeley, in his beautifully illustrated article on the surgery of the colon, gives a very complete and practical summary of diagnosis and treatment. He regards the two-stage methods and that of Devine with favour.

Over 200 pages are occupied by articles on intestinal obstruction and peritonitis by Cokkinis. In addition to the more or less routine description of varieties and types of obstruction there is an excellent discussion of the underlying pathology and physiology of the condition. The summary of treatment of a case of paralytic ileus given in fourteen sentences will appeal to every surgeon who wants simple and clear guidance in dealing with this desperate condition. We think it a pity that one or two coloured illustrations are not given in this section in place of many of the black-and-white figures, which fail to show the condition of congestion or gangrene. A posthumous article by Tyrrell-Gray on paralytic ileus is full of interest, but we are rather surprised that this subject should have been treated twice by different authors. The complications following abdominal operation are dealt with in a very practical way by Maingot.

Part III is occupied entirely by the section on diseases of the anus and rectum by Miles (220 pages), and is wonderfully complete and informative. The description of common ailments, piles, and fistulæ is very complex. The incidence of piles appears to be governed by mathematical laws, and of fistulæ there are seventeen different varieties. The chapters on cancer of the rectum contain convincing arguments in favour of the abdomino-perineal excision. The mortality-rate has been reduced to 10 per cent by the use of spinal percaine with gas and oxygen for anæsthesia, by eliminating patients with reduced cardiac energy, and by the routine use of blood transfusion. Methods by which the anal sphincter can be retained are not mentioned, the use of radium for inoperable cases is not discussed, neither are the operative methods of dealing with intractable pain.

Parts IV and V cover the radiography of the alimentary and urinary tracts (Cecil Bull) with clear radiograms and diagrams of typical conditions; the technique of radium treatment is by Stanford Cade and Malcolm Donaldson. All these articles are very complete and well illustrated.

It remains to give an appreciation of the book as a whole. We can only say that it is very good, and we look forward to the succeeding two volumes with great interest. The days are gone when one man like Erichsen could write a complete text-book of surgery single-handed. The present volume shows wonderful team-work, with the inevitable overlapping and redundancy and conflict of views reduced to a minimum. The due weight given both to the physiological and medical aspects of surgical diseases is quite in accordance with advanced modern teaching. The paper, printing, and illustrations are all good, though we think that a few coloured figures would have been helpful.

We heartily congratulate the Editor and his collaborators on their energy and industry in producing this monumental work, which will soon be regarded as the standard English book of reference.

The Early Diagnosis of the Acute Abdomen. By ZACHARY COPE, B.A., M.D., M.S. (Lond.), F.R.C.S., Surgeon to St. Mary's Hospital, Paddington, etc. Seventh edition. Medium 8vo. Pp. 254 + xiv, with 33 illustrations. 1935. London: Humphrey Milford, Oxford University Press. 10s. 6d. net.

THOUGH we much dislike the term 'acute abdomen', and would vastly prefer the title to have been "The Early Diagnosis of Acute Abdominal Crises", yet we gladly acknowledge this little book to be of the greatest service and undoubtedly the best of its kind. It has now reached its seventh edition—a fact which speaks, literally, 'volumes' for its efficiency. The most important addition is a description, with two figures, of the use of X-rays in the diagnosis of acute intestinal obstruction, a method which is extensively employed in America though only to a small degree as yet in this country. Where, as in hospitals, the requisite apparatus is available, most valuable information can be thus obtained, especially with regard to the extremely important question whether the site of obstruction is in the small or the large intestine. We are glad to note that the author does not describe intussusception under acute intestinal obstruction but allots it a chapter to itself. It is important to recognize that acute intussusception is not primarily an obstruction but rather a 'displacement' or 'dislocation' of the bowel, and that the object of early diagnosis and treatment of this condition is to prevent it from ever becoming an obstruction. We disagree profoundly with the statement on p. 117 that "it is a mistake to treat strangulated external hernia in a different category from obstruction in which no external cause is to be found". It is only by gross carelessness that strangulated hernia can be overlooked even in its earliest stages, so that its mortality should be, and

is actually, much less than that of other cases of obstruction, where, there being no external swelling, the diagnosis and treatment are alike much more difficult. To include strangulated hernia with these latter is to reduce very materially the total mortality, and the same criticism applies to the inclusion of intussusception. Thus, in the largest series of cases of acute intestinal obstruction yet published—that of 6892 cases collected and presented to the Section of Surgery at the Centenary meeting of the British Medical Association in 1932 by Vick—the mortality of all forms together was 26·2 per cent. If strangulated hernia is excluded (whose mortality was 17·8 per cent), the mortality is increased to 38·8 per cent, and if, further, idiopathic intussusception is excluded, the mortality becomes 39·9 per cent. This is the real mortality of those cases of acute intestinal obstruction the cause of which is not readily obvious, and it is this appalling mortality of nearly 40 per cent that clamours for the exercise of every means of early diagnosis: only thus can we hope for its material reduction.

The author rightly deprecates the description—strongly insisted upon elsewhere—of acute appendicitis and acute appendicular obstruction as two separate entities. To stress such distinction is rather apt to lead to the conclusion that cases of acute appendicitis without obstruction do not need immediate operation—a most pernicious doctrine. The severity and frequency of the vomiting at the onset of acute appendicitis indicate, as the author correctly states, the degree of distension of the appendix, and, consequently, the immediate risk of perforation. In Chapter V is an excellent account of the differential diagnosis of acute appendicitis, and the misleading absence of abdominal rigidity in pelvic appendicitis is properly emphasized.

We have never seen a patient "writhing in agony" at the onset of perforation of a gastric or duodenal ulcer; on the contrary, although he may scream in his agony, his muscular efforts are directed to keeping his abdomen as still as possible. The contrast between the abdominal stillness and rigidity of a case of perforated gastric ulcer and the extreme restlessness and abdominal flaccidity of one of rupture of an ectopic pregnancy is most impressive.

Acute pancreatitis receives an excellent description, and emphasis is rightly laid upon the profound degree of shock that usually accompanies the pain, the persistence of the severe pain in the back, and the marked cyanosis of the face and extremities. The last chapter is one of the most useful in the book, and deals with those diseases which simulate acute abdominal crises. The table of differences between acute thoracic inflammations and acute abdominal crises on p. 236 will well repay most careful study.

The book is clearly written, and the simplicity of the illustrations is a fitting accompaniment to the extreme lucidity of the author's method of expression.

The Early Diagnosis of Malignant Disease. By GEOFFREY KEYNES, M.A., M.D. (Cantab.), F.R.C.S., Assistant Surgeon to St. Bartholomew's Hospital, etc. F'cap 8vo. Pp. 70. 1935. London: John Bale, Sons & Danielsson, Ltd. 2s. 6d. net.

THIS book has the merits of brevity, clearness, and low price, and will be very helpful to the general practitioner. It fails, however, to state clearly that the main duty of a practitioner who suspects early cancer is to obtain for the patient an expert opinion. Mr. Keynes thinks the use of the terms 'malignant' and 'malignancy' encourages pessimism. He insists on the fact that really early cancer is local, and therefore often curable, and that whatever progress is made in surgical and radiological technique, the end-results will not improve unless diagnosis becomes earlier and more precise, and patients become more ready to seek early advice. He thinks the practitioner should teach the public that early cancer is painless and often curable. He should be ready to act on suspicion only and should develop an attitude of mind which, if it occurred in the patient, would be a morbid cancer-phobia.

While not blind to its difficulties, the author thinks that an annual general overhaul would be a step towards the earlier detection of cancer. The importance of radiology is stressed, especially in suspected cancer of the stomach and bowel. The various forms of cancer are then considered *seriatim* from a diagnostic point of view.

In a book the general teaching of which is so sound, criticism must be confined to points of detail. It is stated that "Any breast lesion, however small, that is really hard in consistency is probably a cancer." May not a fibro-adenoma or a small cyst be really hard? No teaching is given as to the clinical differences between carcinomatous and mastitic enlargement

of the axillary glands, and nothing is said about the importance of slight flattening of the nipple, or of the retraction of individual duct-orifices upon its surface. One must emphatically dissent from the statement that "In the great majority of breasts affected by mastitis the change is diffuse and more or less uniform." The granular thickenings of chronic mastitis when examined on the flat with two hands are generally partial and sector-shaped, affecting particular lobes or groups of lobes. They are most often felt in the upper and outer quadrant.

The section on gastric cancer is especially good, and is illustrated by X-ray tracings which show that the healing of a chronic ulcer may not prevent subsequent malignancy. The free use of radiological examination in the dyspepsia of middle life is recommended as the only means of early diagnosis. The achlorhydria seen in 65 per cent of cancers is shown to be due to previous chronic gastritis.

Mr. Keynes has fallen a victim to the prevalent fashion of regarding melanotic sarcoma as carcinoma. Surely the work of Ribbert established once for all its sarcomatous nature.

Der Schenkelhalsbruch: ein mechanisches Problem. By Dr. FRIEDRICH PAUWELS (Aachen). Royal 8vo. Pp. 157, with 186 illustrations. 1935. Stuttgart: Ferdinand Enke. Paper covers, RM. 13.60; bound, RM. 15.00.

A VERY excellent monograph on the mechanical factors which are involved in the production, prognosis, and treatment of fractures of the femoral neck. The subject is worked out with mathematical precision accompanied by diagrams and formulæ. Probably this somewhat abstruse presentation will repel rather than attract the average surgeon's attention; but, on the other hand, the diagrams, X-rays, and clinical photographs are clear and convincing. We are accustomed to classify fractures of the femoral neck according to their level as between the head and the trochanter of the femur; but this author demonstrates that it is much more important to group them according to the direction of the fracture. Thus in Grade 1 the fracture is nearly horizontal, in Grade 2 it is about 45° to the horizontal, and in Grade 3 nearly vertical. The important practical considerations are: (1) The liability to non-union is in direct proportion to the verticality of the line of fracture; and (2) That in cases of non-union, the proper treatment is Schanz's osteotomy. In this operation a wedge is removed from below the great trochanter. Pins are inserted above and below the osteotomy, and the femur is angulated. When healing has occurred the adduction of the trochanter will have brought it below the head of the femur, the line of fracture will be horizontal, and union will take place.

BOOK NOTICES

[*The Editorial Committee acknowledge with thanks the receipt of the following volumes. A selection will be made from these for review, precedence being given to new books and to those having the greatest interest for our readers.*]

Traité de Chirurgie d'Urgence (Félix Lejars). By PIERRE BROCQ, Professor agrégé à la Faculté de Médecine de Paris; Chirurgien des Hôpitaux; and ROBERT CHABRUT, Ancien Chef de Clinique à la Faculté de Médecine de Paris. Ninth edition. Large 8vo. One volume of 1286 pages, with 1250 illustrations. Paper covers, Fr. 170; bound, Fr. 190. In two volumes (Pp. 1299 + xv), Fr. 200. 1936. Paris: Masson et Cie.

A Terminology of Operations of the University of Chicago Clinics. By HILGER PERRY JENKINS, M.D. Crown 8vo. Pp. 99 + xx. 1935. U.S.A.: The University of Chicago Press; Great Britain and Ireland: Cambridge University Press. 4s. 6d. net.

Urology in Women: A Handbook of Urinary Diseases in the Female Sex. By E. CATHERINE LEWIS, M.S.(Lond.), F.R.C.S., Surgeon to the Royal Free Hospital; Surgeon and Urologist to the South London Hospital for Women. 9 × 6 in. Pp. 100 + viii, with 31 illustrations. 1936. London: Baillière, Tindall & Cox. 6s. net.

An Index of Differential Diagnosis of Main Symptoms. Edited by HERBERT FRENCH, C.V.O., C.B.E., M.A.(Oxon.), F.R.C.P.(Lond.), Consulting Physician to Guy's Hospital, etc. Super royal 8vo. Pp. 1145 + xii, with 742 illustrations (196 in colour). 1936. Bristol: John Wright & Sons Ltd. 63s. net.

Lehrbuch der Kriegschirurgie. By Dr. CARL FRANZ, Honorarprofessor der Universität, Berlin, etc. Second edition. Large 8vo. Pp. 424 + vii, with 121 illustrations. 1936. Berlin: Julius Springer. Paper covers, RM. 26.60; bound, RM. 28.80.

Zur Erkennung und Begutachtung von Schädelgrundbrüchen. By HANS HELLNER. Part 19 of *Hefte zur Unfallheilkunde*. Large 8vo. Pp. 43, with 17 illustrations. 1936. Berlin: Julius Springer. RM. 4.40.

Brompton Hospital Reports. Volume IV, 1935. Demy 8vo. Pp. 259 + viii. Illustrated. London: The Secretary, The Hospital for Consumption, Brompton, S.W.3. 2s. 9d.

Das Hämorrhoidalleiden: seine Komplikationen und deren Behandlung. By Dr. KASPER BLOND and Dr. HERBERT HOFF. With a Foreword by Prof. Dr. LEOPOLD SCHÖNBAUER. Large 8vo. Pp. 121 + viii, with 50 illustrations. 1936. Leipzig and Vienna: Franz Deuticke. Paper covers, M. 12; bound, M. 14.40.

Mechanics of Normal and Pathological Locomotion in Man. By ARTHUR STEINDLER, M.D., F.A.C.S., Professor of Orthopedic Surgery, the State University of Iowa. Large 8vo. Pp. 424 + xviii, with 453 illustrations. 1936. London: Baillière, Tindall & Cox. 36s. net.

Handbook of Surgery. By ERIC C. MEKIE, M.B., Ch.B., F.R.C.S.E., Medical Officer, Malayan Medical Service. With a Foreword by JOHN FRASER, M.C., M.D., Ch.M., F.R.C.S.E., Regius Professor of Clinical Surgery, University of Edinburgh. Crown 8vo. Pp. 699 + xii, with 24 illustrations. 1936. Edinburgh: E. & S. Livingstone. 12s. 6d. net.

Zweite internationale Kropfkonferenz (Bern, Aug. 10–12, 1933). Edited by Dr. OTTO STINER. Large 8vo. Pp. 698 + li. Illustrated. 1935. Bern: Hans Huber. Sv. Fr. 25.

Tecknik und Ergebnisse der Gaumenplastik. By Dr. GEORG AXHAUSEN (Berlin), Large 8vo. Pp. 126, with 176 illustrations. 1936. Leipzig: Georg Thieme. Paper covers, M. 17; bound, M. 18.

Emergency Surgery. By HAMILTON BAILEY, F.R.C.S., Surgeon, Royal Northern Hospital, London, etc. Second edition. Demy 8vo. Pp. 842 + x, with 812 illustrations. 1936. Bristol: John Wright & Sons Ltd. 50s. net.

An Index of Treatment. Edited by ROBERT HUTCHISON, M.D., LL.D., F.R.C.P., Consulting Physician, London Hospital, and Hospital for Sick Children, Great Ormond Street. Eleventh edition. Super royal 8vo. Pp. 1020 + xv. Illustrated. 1936. Bristol: John Wright & Sons Ltd. 42s. net.

The Human Foot: its Evolution, Physiology and Functional Disorders. By DUDLEY J. MORTON, Associate Professor of Anatomy, College of Physicians and Surgeons, Columbia University. Large 8vo. Pp. 244 + xvi, with 100 illustrations. 1935. New York: Columbia University Press. London: Oxford University Press (Humphrey Milford). 15s. net.

Surgical Emergencies in Children. By HAROLD CLIFFORD EDWARDS, M.S.(Lond.), F.R.C.S., Surgeon and Lecturer in Surgery, King's College Hospital, London, etc. Demy 8vo. Pp. 274 + vii, with 99 illustrations. 1936. London: Baillière, Tindall & Cox. 12s. 6d. net.

Diagnostic Roentgenology. Edited by ROSS GOLDEN, M.D., Professor of Radiology, the College of Physicians and Surgeons, Columbia University; Director of the Department of Radiology, Presbyterian Hospital, New York. $9\frac{1}{2} \times 7\frac{1}{4}$ in. Pp. 867 + xi, with 964 illustrations. 1936. New York and Edinburgh: Thomas Nelson & Sons. $20.00.

Les petites Règles de la Chirurgie parfaite. By J. OKINCZYC, Professeur agrégé; Chirurgien des Hôpitaux de Paris. Royal 8vo. Pp. 60. 1936. Paris: Masson et Cie. Fr. 12.

The Study of Anatomy. Written for the Medical Student. By S. E. WHITNALL, M.A., M.D., B.Ch.(Oxon.), M.R.C.S., L.R.C.P., F.R.S.(Canada), Professor of Anatomy in the University of Bristol, 1935. Third edition. Crown 8vo. Pp. 113. 1936. London: Edward Arnold & Co. 4s. 6d. net.

Surgical Diseases and Injuries of the Genito-Urinary Organs. By Sir JOHN THOMSON-WALKER, D.L., M.B., C.M.Ed., F.R.C.S.Eng., Consulting Urologist and Emeritus Lecturer in Urology, King's College Hospital, etc. Second edition, revised: edited by KENNETH WALKER, M.A., M.B., B.C.(Cantab.), F.R.C.S..Eng., Jacksonian Prizeman and Hunterian Professor, R.C.S., 1911, 1922, 1924, 1933; Lecturer in Venereal Diseases, St. Bartholomew's Hospital; etc. Medium 8vo. Pp. 974 + xviii, with 283 illustrations and 25 colour and 33 black-and-white plates. 1936. Cassell & Co. Ltd. 32s. 6d. net.

A Synopsis of Surgical Anatomy. By ALEXANDER LEE MCGREGOR, M.Ch. (Edin.), F.R.C.S. (Eng.), Lecturer on Surgical Anatomy, University of Witwatersrand, etc. With a Foreword by Sir HAROLD J. STILES, K.B.E., F.R.C.S.E. Third edition. Crown 8vo. Pp. 664 + xviii, with 648 illustrations. 1936. Bristol: John Wright & Sons Ltd. 17s. 6d. net.

Ueber das Zwölffingerdarmgeschwür. By Prof.-Dr. P. CLAIRMONT (Zurich). Part 7 of *Vorträge aus der praktischen Chirurgie*. Royal 8vo. Pp. 29. 1936. Stuttgart: Ferdinand Enke. RM. 1.80.

A Textbook of Surgery. By American Authors. Edited by FREDERICK CHRISTOPHER, B.S., M.D., F.A.C.S., Associate Professor of Surgery at Northwestern University Medical School, etc. Royal 8vo. Pp. 1608+xix, with 729 illustrations. 1936. Philadelphia and London: W. B. Saunders Co. 42s.

Baillière's Synthetic Anatomy. By J. E. CHEESMAN. Part XIII. The Eye and Orbit. Plates 7½×9 in. 1936. London: Baillière, Tindall & Cox. 3s. net.

A Textbook of Roentgenology. By BEDE J. MICHAEL HARRISON, M.B., CH.M., D.M.R.E. (Cantab.), F.A.C.R., Director of Department of Roentgenology, Vancouver General Hospital, etc. Medium 8vo. Pp. 826+xxvi, with 238 illustrations. 1936. London: Baillière, Tindall & Cox. 45s.

The Life and Works of Charles Barrett Lockwood (1856–1914). By ERIC C. O. JEWSBURY, M.A., B.M., B.CH. (Oxon.). The Wix Prize Essay, St. Bartholomew's Hospital, 1934. Crown 8vo. Pp. 104+viii, with 12 illustrations. 1936. London: H. K. Lewis & Co., Ltd. Cloth, 3s. 6d. net; paper covers, 2s. 6d. net.

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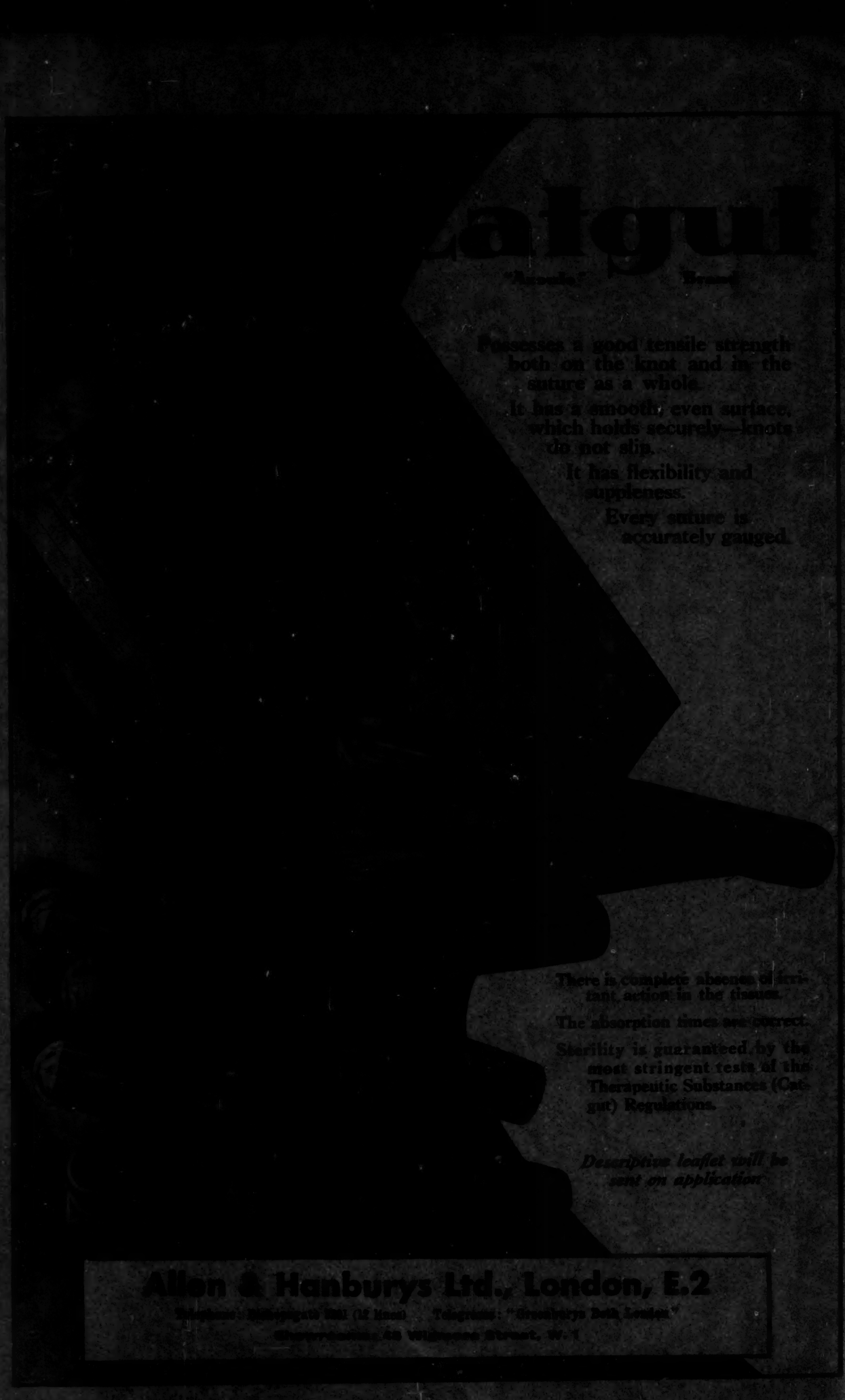

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